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13. ABSTRACT *(Maximum 200 words)* This study examined the efficacy of hyperhydration approaches during compensable and uncompensable exercise-heat stress and the impact of hyperhydration on physiologic response and tolerance to heat strain. The general approach was to determine if 1-h pre-exercise hyperhydration (29.1 ml·kg LBM^{-1} with or without glycerol 1.2 g·kg LBM^{-1}) provided a physiologic advantage. During compensable heat stress (CHS) the evaporative heat loss required (E_{req} = 293 W·m^{-2}) to maintain steady-state core temperature was less than the maximal capacity (E_{max} = 462 W·m^{-2}) of the climate for evaporative heat loss (E_{req}/E_{max} = 63%). During uncompensable heat stress (UCHS) the E_{req} (366 W·m^{-2}) was greater than E_{max} (88 W·m^{-2}; E_{req}/E_{max} = 416%) and core temperature continued to rise until exhaustion from the heat strain occurred. Eight heat-acclimated men completed 5 trials (euhydration, glycerol hyperhydration, and water hyperhydration both with and without rehydration (replace fluid lost during exercise) in CHS and 3 trials (control , glycerol hyperhydration and water hyperhydration) in UCHS. During exercise in the heat (35°C, 45% rh) there was no difference between hyperhydration methods for increasing total body water. During CHS, hyperhydration did not alter core temperature, skin temperature, whole body sweating rate, local sweating rate, sweating threshold temperature, sweating sensitivity, or heart rate responses compared to euhydration trial. Likewise, no difference was found between water and glycerol hyperhydration for these physiologic responses. During UCHS, hyperhydration did not alter thermal or cardiovascular (stroke volume, cardiac output, blood pressure, total peripheral resistance) responses or heat strain tolerance compared to the control trial. In addition, it was observed that glycerol solution ingestion often results in nausea and headaches. It is concluded that hyperhydration provides no meaningful advantages over the maintenance of euhydration during exercise-heat stress.

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HYPERHYDRATION: PHYSIOLOGIC AND THERMOREGULATORY EFFECTS DURING COMPENSABLE AND UNCOMPENSABLE EXERCISE-HEAT STRESS

by

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May 1996

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EXECUTIVE SUMMARY

This study examined the efficacy of hyperhydration approaches during compensable and uncompensable exercise-heat stress and the impact of hyperhydration on physiologic response and tolerance to heat strain. The general approach was to determine if 1-h pre-exercise hyperhydration (29.1 ml·kg LBM^{-1} with or without glycerol 1.2 g·kg LBM^{-1}) provided a physiologic advantage. During compensable heat stress (CHS) the evaporative heat loss required (E_{req} = 293 $W \cdot m^{-2}$) to maintain steady-state core temperature was less than the maximal capacity (E_{max} = 462 $W \cdot m^{-2}$) of the climate for evaporative heat loss (E_{req}/E_{max} = 63%). During uncompensable heat stress (UCHS) the E_{req} (366 $W \cdot m^{-2}$) was greater than E_{max} (88 $W \cdot m^{-2}$; E_{req}/E_{max}= 416%) and core temperature continued to rise until exhaustion from the heat strain occurred. Eight heat-acclimated men completed 5 trials (euhydration, glycerol hyperhydration, and water hyperhydration both with and without rehydration (replace fluid lost during exercise) in CHS and 3 trials (control , glycerol hyperhydration and water hyperhydration) in UCHS. During exercise in the heat (35ºC, 45% rh) there was no difference between hyperhydration methods for increasing total body water. During CHS, hyperhydration did not alter core temperature, skin temperature, whole body sweating rate, local sweating rate, sweating threshold temperature, sweating sensitivity, or heart rate responses compared to euhydration trial. Likewise, no difference was found between water and glycerol hyperhydration for these physiologic responses. During UCHS, hyperhydration did not alter thermal or cardiovascular (stroke volume, cardiac output, blood pressure, total peripheral resistance) responses or heat strain tolerance compared to the control trial. In addition, it was observed that glycerol solution ingestion often results in nausea and headaches. It is concluded that hyperhydration provides no meaningful advantages over the maintenance of euhydration during exercise-heat stress.

INTRODUCTION

Hydration (body water) status is important for temperature regulation and exercise performance in hot environments. During exercise in hot environments, sweating rates often exceed 1.0 $L \cdot h^{-1}$ (2,7) and have been reported as high as 3.7 $L \cdot h^{-1}$ (4). Therefore, prolonged exercise without rehydration results in a considerable loss of body water. Hypohydration (reduced total body water) results in elevated deep body temperatures (1,53,78,113), reduced heat tolerance (106) and reduced aerobic exercise performance (9,80,114,121). Several investigators have reported that the magnitude of additional heat strain is linearly related to the hypohydration level during exercise-heat stress (36,41,65,106). It is not clear what effect hyperhydration (increased total body water) has on thermoregulation and tolerance to exercise-heat stress (106). This study will examine the effects of hyperhydration on thermoregulation and heat tolerance during both compensable and uncompensable heat stress.

Hyperhydration, or overdrinking, has been suggested to improve, above euhydration levels, thermoregulation and exercise-heat performance (55,61,69); however, its efficacy has not been systematically evaluated. Few studies have reported the effects of hyperhydration on performance. One study reported no ergogenic advantage (41); several studies report that hyperhydration can reduce thermal strain during exercise-heat stress (61,69); while several other studies indicate no thermoregulatory advantage (41,72). These conflicting results may be due to differences in experimental design (e.g., inadequate controls, climatic conditions, subject population) and in methods used to achieve hyperhydration (41,61,69,72). If hyperhydration does improve exercise-heat performance, it might be through an improved ability to dissipate body heat (i.e., through elevated sweating or skin blood flow), reduced cardiovascular strain (i.e., improved maintenance of cardiac stroke volume), and/or improved abilities to tolerate thermal strain.

The physiologic mechanisms of improved body heat dissipation and decreased cardiovascular strain from hyperhydration would be an expanded blood volume and attenuated plasma hypertonicity (79,93,97). Studies that have directly expanded blood volume (e.g., infusion) have usually reported decreased cardiovascular strain (47,76,100) during exercise, but also reported disparate results concerning improved

heat dissipation (19,76,107) and exercise-heat performance (19,60). Studies that have attenuated plasma hyperosmolality during exercise-heat stress generally show improved heat dissipation (27,28,44,97), but have not addressed exercise performance.

Hyperhydration could improve exercise-heat performance by delaying the development of hypohydration. On the other hand, if hyperhydration substantially increases sweating rates (61,69), then hyperhydration would result in a more rapid dehydration. This could minimize or negate any physiological advantage gained if fluids are not replaced. For example, Lyons and colleagues (61) reported that glycerol/water hyperhydration elevated the sweating rate by ~430 $ml \cdot h^{-1}$ above the control during the last 30 min of exercise-heat stress. If hyperhydration elevates sweating rates by such substantial amounts, then it will be critical to maintain drinking, to avoid hypohydration, during the subsequent exercise-heat exposure. If hyperhydration does not substantially elevate sweating rate, then it could be used to improve exercise-heat performance when additional fluids are not available.

Soldiers on a chemical battlefield, firefighters and foundry workers are sometimes exposed to uncompensable heat stress. This occurs when they work in oppressively hot and/or humid environments (83,84) or work while wearing protective clothing (54). During uncompensable heat stress the body's evaporative cooling requirement exceeds the climate's cooling capacity (8,58,59). In compensable heat stress environments, thermoregulatory control can compensate for increased body heat storage and maintain an elevated steady-state core temperature; whereas, in uncompensable heat stress the thermoregulatory system is unable to compensate and core temperature continues to rise. So in an uncompensable heat stress environment any improved physiological heat dissipation capability (mediated by hyperhydration) would be with little or no thermal benefit. Uncompensable heat stress is associated with heat exhaustion occurring at relatively low core temperatures (67,106), because the displacement of blood to the skin (via increased cutaneous dilation and compliance) causes marked cardiovascular strain and instability (11,89,94).

During uncompensable heat stress conditions, a hyperhydration method that defends or expands blood volume for extended periods should, theoretically, decrease cardiovascular strain and improve exercise-heat performance. The ergogenic effects of hyperhydration have not been evaluated during uncompensable heat stress conditions.

Ideally, hyperhydration should increase total body water for extended periods (at least several hours) and allow plasma volume to be maintained or expanded. Riedesel et al. (81) and our laboratory (30) have recently demonstrated that glycerol/water hyperhydration will increase total body water with no increase in plasma volume for prolonged durations. These findings suggest that with glycerol hyperhydration the additional water is distributed to both intra- and extracellular compartments so that it might be able to provide a fluid reservoir to help defend plasma volume during exercise-heat stress.

OBJECTIVES

The purpose of this study is to determine the efficacy of hyperhydration for improving thermoregulation and physiological tolerance to exercise-heat stress. The study will consist of two phases: Phase I will examine thermoregulatory effects of hyperhydration during compensable exercise-heat stress. Glycerol hyperhydration will be compared to water hyperhydration and euhydration under conditions of maintained hydration and progressive dehydration. Phase II will evaluate the physiological tolerance effects of hyperhydration during uncompensable exercise-heat stress. Glycerol hyperhydration will be compared to water hyperhydration and euhydration during progressive dehydration. These experiments will systematically examine possible heat strain reduction-ergogenic benefits of glycerol hyperhydration for a variety of conditions (compensable, uncompensable heat stress; rehydration vs. nonrehydration) that have military and civilian importance. In addition, these experiments will delineate the physiological mechanisms responsible for any thermoregulatory, heat tolerance or exercise performance effects.

SPECIFIC QUESTIONS

1. Does hyperhydration reduce heat strain during compensable heat stress or increase physiological tolerance during uncompensable heat stress?
2. If rehydration is not continued during exercise-heat stress, will the ergogenic advantages of hyperhydration be maintained?

3. Is glycerol hyperhydration more effective than water hyperhydration in mediating reduced heat strain, increased physiological tolerance and improved performance during exercise-heat stress?
4. By what physiological mechanism does hyperhydration reduce heat strain, increase physiological tolerance and improve exercise performance during uncompensable and compensable heat stress? Does hyperhydration improve thermoregulatory control evidenced by a reduced threshold and increased sensitivity of sweating during compensable heat stress? Does hyperhydration increase physiological tolerance as evidenced by improved cardiovascular stability (maintaining cardiac output, stroke volume and blood pressure) during uncompensable heat stress?

HYPOTHESES

1. Hyperhydration will reduce heat strain during compensable exercise-heat stress. This thermal advantage will be mediated by improved sweating responses such as reduced threshold temperature and increased sensitivity.
2. Hyperhydration will improve physiological tolerance to heat strain during uncompensable exercise-heat stress. This tolerance advantage will be mediated by improved cardiovascular stability.
3. Hyperhydration with glycerol will be more effective than hyperhydration with only water in mediating reduced heat strain and improved performance during exercise-heat stress.
4. Hyperhydration ergogenic effects will be more evident with continued fluid replacement during compensable exercise-heat stress.

BACKGROUND

THERMOREGULATION

During rest and exercise, body heat is exchanged with the environment by conduction, convection, radiation and evaporation (33). The body can gain or lose heat by these mechanisms, depending on the relative temperatures of the skin and climate. When ambient temperature exceeds skin temperature, the body will gain heat from the

environment. Evaporation of sweat is the major mechanism in which heat will be lost when ambient temperature is high. Heat lost by evaporation is dependent on the water vapor pressure gradient between the skin and the surrounding ambient air. The greater the water vapor gradient, the greater is the potential for evaporative cooling. Evaporation of one gram of water from the skin will result in ~ 2.45 kJ heat loss (33). Since the mechanical work is minimal during treadmill exercise, most of the metabolic rate is converted to body heat. So, an individual on a treadmill with a metabolic rate of 2 L $O_2 \cdot min^{-1}$ (695 W) would need to dissipate 695 W (0.695 kJ $\cdot s^{-1}$ or 41.7 kJ $\cdot$ min^{-1}) of heat to remain at a steady-state body temperature. Specific heat of body tissues is ~ 3.5 kJ $\cdot kg^{-1} \cdot °C^{-1}$ (57,95); so, a 75 kg person has a heat capacity of (3.5 kJ$\cdot kg^{-1} \cdot °C^{-1} \cdot$ 75 kg) ~262 kJ $\cdot °C^{-1}$. If this person exercised in a climate with no dry heat loss and no evaporation, body temperature would rise 1 °C every ~ 6 min (262 kJ $\cdot °C^{-1} \div$ 41.7 kJ $\cdot min^{-1}$). To maintain a steady-state body temperature this person would need to evaporate 17 g (41.7 kJ$\cdot min^{-1} \div$ 2.45 kJ $\cdot$ g $sweat^{-1}$) of sweat every minute.

HYDRATION AND EXERCISE-HEAT STRESS

Numerous studies have examined the effects of hydration on thermoregulatory and cardiovascular responses during exercise stress. As early as 1944, Pitts and associates (78) demonstrated that if fluids were restricted during prolonged exercise, core temperature would continue to rise. If, however, the fluids lost were replaced during the exercise, the subjects achieved steady-state core temperatures. Likewise, in 1947, Rothstein and Towbin (86) reported that when fluids were restricted in subjects exposed to extreme heat (i.e., 50°C), both core temperature and heart rate were elevated when compared to trials in which body weight was maintained by fluid replacement. Similar observations have since been reported (97).

Exercise-heat stress may be compromised by the cardiovascular system's inability to provide enough blood to working skeletal muscles, as well as to the skin for heat dissipation (49,97,98). The cardiovascular challenge becomes greater when blood volume is reduced because of increased sweating rates and loss of body fluids that are not replaced. Blood volume reduction and increased blood pooling in the cutaneous vasculature during exercise-heat stress can reduce cardiac filling and stroke volume

(89). An increased cardiac contractility may not compensate for the reduced cardiac filling during intense exercise-heat stress and cardiac output may not be maintained (65,92,101).

Increased total body water provides a greater reservoir of fluid, which might be available to redistribute to the vascular spaces, so that cardiac output as well as muscle and skin blood flow can be maintained during exercise-heat stress. A better defense of blood volume is likely to provide a larger fluid reserve for sweat and/or increased cardiovascular stability to support high cutaneous blood flow. However, several studies that increased blood volume have indicated no differences in core temperature (26,64), sweat rate, or cutaneous blood flow (26). The small increase in blood volume that would be expected with hyperhydration should have little impact on performance or thermoregulation, but if hyperhydration can help maintain blood volume (at control levels) during exercise-heat stress then this impact should become more evident during exercise-heat stress.

It is known that humans drinking ad libitum do not drink sufficient fluid during exercise-heat stress (1,2,78). It is recommended that persons in these situations drink despite lack of thirst (97,98). Any thermoregulatory advantage gained by heat acclimation or high aerobic fitness may be lost during exercise-heat stress by hypohydration (14,15,103). The adverse effects of hypohydration on exercise performance may be due to an impairment of dry and/or evaporative heat loss mechanisms, as well as a reduced tolerance to heat strain (exhaustion from heat strain occurring at a lower core temperature) (67,106). The negative effects of hypohydration have been well documented, but the effects of hyperhydration during exercise-heat stress are inconclusive and need further study (102).

APPROACHES TO HYPERHYDRATION

Different approaches to achieve hyperhydration in humans have been tried. Hyperhydration approaches have included drinking large volumes of fluid, treatment with antidiuretic hormone (ADH) followed by increased fluid consumption (72) and, more recently, the use of glycerol solutions (30,61,81). These various methods have had limited success in the amount of fluid retained. The problem is that excess fluid is

excreted rapidly; therefore, increases in body water are transient. Consumption of hypotonic (relative to plasma) fluid will reduce the osmolality in plasma (56,112). Osmoreceptors sense the hypotonic plasma and reduce production and release of ADH (vasopressin). A decrease in plasma ADH will reduce renal collecting duct permeability to water and result in an increase urine flow (82). Also, any factor that results in an increased cardiac atrial stretch such as fluid overload can increase atrial natriuretic factor release (120) (ANF, found mainly in the cardiac atrium). Atrial natriuretic factor stimulates increased glomerular filtration and free water clearance, and inhibits ADH release. Both ADH and ANF are important hormones that regulate fluid balance (29) and limit achievable hyperhydration level (10,82,119,120).

One approach to achieve hyperhydration has been through the administration of ADH; the theory being that if ADH is administered prior to hyperhydration, urine flow would decrease and more fluid would be retained. Nadel et al. (72) administered ADH using either lysine-8-vasopressin nasal spray (8 U) or aqueous arginine vasopressin (0.75 U) injected subcutaneously. Ten minutes post administration the subjects drank 2 L of water to attain hyperhydration. The amount of ADH given was reported to reduce urine flow to about 30% of the control for 3-6 h. Plasma volume increased 1% to 3% based on hematocrit and hemoglobin values. Unfortunately, they did not report body mass changes or how much water was retained over the experiment.

Riedesel et al. (81) tested glycerol's effectiveness to promote hyperhydration. They had subjects ingest 0.1% NaCl solution, 21.4 $ml \cdot kg^{-1}$ in 40 min either with or without glycerol (control trial). Subjects were divided into three groups based on glycerol dosage (0.5, 1.0 or 1.5 g $glycerol \cdot kg^{-1}$). They reported approximately 40% (600 ml) of the fluid was retained after 200 min in the two higher glycerol dosage groups, and approximately 30% was retained in the 0.5 g $glycerol \cdot kg^{-1}$ dosage group, while less than 20% of the fluid was retained in the control group. They reported no change in plasma volume with hyperhydration. However, since only 7.5% of total body water (TBW) is in plasma, if TBW increased 600 ml, plasma volume should only increase by ~ 45 ml (if it were equally distributed between intracellular and extracellular fluid). Thus the change might have exceeded the measurement's resolution. Renal data including glomerular filtration rate, osmotic clearance, free water clearance and fluid regulating endocrine hormones were not measured making it difficult to determine mechanisms responsible for increased total body water following hyperhydration with glycerol.

Recently Freund and colleagues (30) studied the physiological responses of glycerol ingestion with water in an attempt to confirm glycerol's effectiveness for hyperhydration and to determine the mechanisms responsible for glycerol's actions. In each trial their subjects ingested fluid (37 $ml \cdot L^{-1}$ TBW); in one trial the fluid was only water and in the other trial it was water and glycerol (1.5 $g \cdot L^{-1}$ TBW). They reported 60% of the water was retained after 180 min in the glycerol trial as compared to 32% for the water only trial. Higher plasma osmolality and ADH levels in the glycerol trial were reported. The differences in ADH corresponded to times at which urine flow rate and free water clearance were lower in the glycerol trial, indicating that ADH may have contributed to glycerol's effectiveness for fluid retention (30). Another mechanism for glycerol's effectiveness in water retention was reported to be a direct effect of glycerol on the kidney. Freund et al. (30) postulated that glycerol's effectiveness is related directly to increasing the kidney's medullary osmotic concentration gradient and thereby increasing water reabsorption and reducing diuresis.

Both ADH and glycerol taken with large volumes of fluid will increase fluid retention. The mechanisms by which they increase water retention are different. ADH acts on the renal collecting ducts to cause increased reabsorption of solute free water (82). For the renal effects of ADH to be manifested (i.e., a decrease in urine flow and free water clearance), the experimental design must allow a sufficient period after ADH elevation for renal effects to be apparent. Glycerol may also affect ADH release because of increased plasma osmolality from high plasma glycerol. Several investigators (37,57,62,81) acknowledge that glycerol is evenly distributed between fluid compartments, and any fluid ingested will equilibrate between the intracellular and extracellular fluid compartments. Therefore, it is anticipated that total body water expansion following glycerol ingestion should be reflected by an increase in both intracellular and extracellular fluid compartments (30).

HYPERHYDRATION - EFFECTS ON THERMOREGULATION

Several studies have investigated the effects of hyperhydration on thermoregulation (17,35,41,42,61,69,72,73,75) and exercise performance (9). **Table 1** provides a chronological review of studies evaluating hyperhydration effects on thermoregulation. Briefly, six of the eleven studies observed smaller core temperature

increases during exercise with hyperhydration. Moroff and Bass (69) observed hyperhydration resulted in reductions in core temperature of 0.3°C. Nielsen et al. (73,75) observed similar results in cool and warm environments. Three (61,69,73) of the nine studies examining sweating rate observed higher rates during exercise with hyperhydration. In all cases in which heart rate was reported, heart rate was lower in the hyperhydration experiment during exercise than in the control experiment. Together, these studies support the notion that hyperhydration might provide a thermoregulatory benefit; however, other studies (17,41,72) report no thermoregulatory advantage with hyperhydration during exercise. In addition, the control condition for many of these studies does not represent euhydration, as the subjects were allowed to dehydrate during exercise. This section will systematically review the studies focusing on the effects of hyperhydration during exercise.

Blyth and Burt (9) were the first to report the effects of hyperhydration on performance during exercise-heat stress. Their subjects ran (3.13 $m \cdot s^{-1}$, 8.6% grade) to exhaustion in a hot climate (49°C) following ad libitum fluid intake, and 30 min after drinking 1 L of isotonic saline and 1 L of water. Exhaustion time was not different between the hyperhydration (17.3 min) and control (16.9 min) trials. They also reported no difference in core (rectal) temperature or water loss between trials. But, when they analyzed only the performances of "athletes," they reported the endurance times to be greater in the hyperhydration trial compared to the control, 18.9 min and 17.1 min, respectively, and reported no differences in changes of core temperature or water loss between the trials. Since final core temperatures were not reported, we are unable to determine if hyperhydration affected tolerance to heat strain.

Moroff and Bass (69) were the first to report the effects of hyperhydration on thermoregulation during exercise-heat stress. They employed a crossover design in which unacclimated subjects in one trial drank 2 L water (24 °C) in a 50-min period and in another trial drank nothing before exercise (1.56 $m \cdot s^{-1}$, 0% grade). In both trials, during the 90-min exercise-heat stress (49/27 °C dry bulb/wet bulb, wind speed 1.3 $m \cdot s^{-1}$), the 30 subjects were given 300 ml to drink at 20, 40, 60 and 80 min of exercise.

Table 1. Summary of Studies: Hyperhydration Effects on Thermoregulation

Study	Year	Pre-Exercise Hydration Treatments	Exercise-Environmental Conditions	Temperature Core	Temperature Skin	Sweat Rate
Blyth & Burt [9]	1961	2 L 0.45% saline vs ad libitum	run to exhaustion (Ta=49°C)	NC	-	NC
Moroff & Bass [69]	1965	2 L water vs no water	90 min treadmill (Ta=49°C)	↓(0.3°C)	-	↑
Greenleaf & Castle [41]	1971	2.5-3.0 L water vs ad libitum	70 min cycle (~50%$\dot{V}O_2$max) (Ta=24°C)	NC	NC	NC
Nielsen et al. [75]	1971	1.5 L water vs no water or 1.0 L 2% saline	60 min cycle (~50%$\dot{V}O_2$max) (Ta=20°C)	↓(0.5°C) ↑(0.3°C)	- -	- -
Nielsen [73]	1974	1.5 L water vs no water vs 1.5 L 2-3% saline	60 min cycle (45%$\dot{V}O_2$max) (Ta=30°C)	↓(?) ↑(?)	- -	↑ ↓
Gisolfi & Copping [35]	1974	1 L water vs no water vs 1 L with rehydration	120 min treadmill (75%$\dot{V}O_2$max) (Ta=33°C)	↓(0.2°C) ↓(0.8°C)	- -	NC NC
Nadel et al. [72]	1980	2.0 L water with ADH vs euhydration	30 min cycle (55%$\dot{V}O_2$max) (Ta=35°C)	NC	-	-
Grucza et al. [42]	1987	2.0 L water vs euhydration	45 min cycle (~52%$\dot{V}O_2$max) (Ta=23°C)	↓(0.4°C) ↓(△0.2°C)	NC	↓
Candas et al.[17]	1988	0.5 L isotonic solution vs euhydration	4h intermittent cycle (70 W) (Ta=36°C)	NC	NC	NC
Lyons et al.[61]	1990	1.5 L water vs 1.5 L water with glycerol or ad libitum	90 min treadmill (~60%$\dot{V}O_2$max) (Ta=42°C)	↓(0.7°C) NC	- -	↑ NC
Montner et al.[68]	1996	1.7 L water vs 1.7 L with glycerol	bike to exhaustion (74%$\dot{V}O_2$max) (Ta=24°C)	NC	-	NC

NC= No change from control trial. T_a=ambient temperature.

Core (rectal) temperature and heart rate responses were lower after the hyperhydration period and remained lower during the exercise-heat stress. The specific heat capacity of water is 4.18 kJ · kg^{-1} · $°C^{-1}$ and could decrease body temperature (48). Raising the water temperature from 24°C to body temperature (37°C) requires 109 kJ (4.18 kJ· kg^{-1} · $°C^{-1}$ · 2 kg · 13°C); this heat capacity of water theoretically would lower body temperature by ~0.4 °C in a 70 kg person. Since the core temperature difference was reported after drinking, it is likely the ingestion of cold water accounted for some of the difference in body temperature. During the hyperhydration trial, higher sweating rates and greater urine outputs were reported, however, a greater body weight loss was reported in the control trial (1.2% vs. 0.6%). The weight loss indicates hypohydration in the control trial, and a 1.2% hypohydration level accounts for higher core temperatures and heart rates, and lower sweating rates in the control trial.

Moroff and Bass (69) also examined the effect of repeated hyperhydration during exercise-heat stress on the pattern of heat acclimation. Twelve subjects were tested (similar tests as the previous experiment) on day 1 and then divided into two matched groups based on body size and physiologic responses. The volunteers were heat acclimated by walking 100 min at 1.55 $m·s^{-1}$, 0% grade for 9 days (day 2 through day 10). Each group drank 1.5 L during the exercise-heat exposure; the hyperhydration group drank an additional 2.0 L prior to exercise-heat stress each day. The groups were again tested on days 11 and 12. On day 11 neither group was prehydrated, on day 12 both groups were hyperhydrated, and on both days each group drank 1.5 L during the exercise trial. They reported both groups had a significantly greater sweating rate, lower heart rate and lower core temperature when hyperhydrated (day 12) as compared to the control (day 11) conditions. They did not report body weights or if euhydration was maintained during exercise in the control trial. The information presented on the test days would indicate that hyperhydration in these studies resulted in a greater sweat rate, lower heart rate and lower core (rectal) temperature as compared to the control experiments, and acclimation to heat did not alter these observations. The core temperatures were lower after drinking, so, as mentioned in the previous paragraph the lower core temperature could be attributed to the cold water drink in the hyperhydration trial. From the information presented for the acclimation trials, it can not be determined whether differences might have been due to a hypohydration effect.

Greenleaf and Castle (41) studied the effects of water hyperhydration on thermoregulation and exercise performance in a temperate environment. Eight partially heat-acclimated subjects performed three trials. In the first trial, subjects drank 40 $ml \cdot kg^{-1}$(2.5-3 L) of water (37°C) during the hour before exercise; in the second trial (ad libitum), subjects drank 100-200 ml during the hour before exercise, and in the third trial, the subjects were hypohydrated by 5% of their body weight the night prior to the exercise test. In each trial, hydration was maintained at the pre-exercise level by giving subjects fluid every 10 min during the exercise test. The 70-min cycle ergometer exercise (49%$\dot{V}O_2max$) was conducted in a controlled temperate (24°C, 50% rh) climate. The investigators reported no significant differences in core (rectal) temperature or sweating rate during exercise in the hyperhydration as compared to the ad libitum trials. They suggested that during hyperhydration there was a smaller increase in core temperature and a greater sweating rate, but their table does not indicate significant differences. Unfortunately, before starting the ad libitum trials, the subjects were ~1.6% dehydrated.

Nielsen and colleagues (75) studied the effects of hydration on thermoregulation and exercise performance. Their subjects attempted three trials in which they exercised (50%$\dot{V}O_2max$) on a cycle ergometer for 60 min in a temperate (20°C) climate. The three trials were conducted after subjects drank the following: 1) 1.5 L water, 2) 1 L of 2% saline solution, and 3) ad libitum. Nielsen et al. (75) reported lower core (rectal) temperatures and heart rates during exercise when subjects were hyperhydrated with 1.5 liters of water as compared to the control group (no water). They also reported elevated core temperatures when hyperhydrated with a hypertonic saline solution (2% NaCl) as compared to the control conditions. Unfortunately, the investigators did not report detailed data regarding the hydration levels achieved or use of any statistical analyses. The control group did not drink during exercise, so the comparison of hyperhydration to the control group most likely reflects the effects of hypohydration.

Nielsen (73) published a similar study in which the purpose was to determine whether plasma osmolarity affected sweat gland function peripherally or centrally. They had two subjects exercise (45%$\dot{V}O_2max$) for 60 min in two ambient temperatures (7°C and 30°C) on four occasions: 1) 30 min after drinking 1.5 L water, 2) 2-3 hours after drinking 1.5 L of 2-3% NaCl solution, 3) when hypohydrated, and 4) their "normal" hydration condition. They also reported that "in some experiments ADH (Vasopressin

spray, Sandoz. 1 ml 50 IU Lysin vasopressin synth.) was sprayed twice into the nose to prolong hyperhydration." Nielsen stated:

> "The results of the present study are in agreement with our earlier findings (75) that the level of body temperature reached during exercise is related to plasma osmolarity: when plasma osmolarity is increased by intake of 2% or 3% NaCl solution or by dehydration, the level of temperature is higher than when plasma osmolarity is decreased by intake of water."

Sweating rate was greatest in the hyperhydration with water trials. Problems with this study include the use of few subjects, the lack of reported statistical analysis, and the lack of detail regarding the hydration levels achieved or the experimental procedures. The "normal hydration" condition is not a good control group for determining the effects of hyperhydration on thermoregulation; this group did not drink during exercise, so the comparison of hyperhydration to the control group most likely reflects the effects of hypohydration.

Gisolfi and Copping (35) examined the effects of hyperhydration with different fluid replacement regiments on thermoregulation during a 120-min treadmill exercise (75%$\dot{V}O_2$max) in the heat (33.5°/21.5°C db/wb). Five trained subjects performed six trials pertinent to this review, three in which they were hyperhydrated by drinking one L water 30 min prior to beginning exercise and three in which they were euhydrated prior to exercise. During exercise the subjects were given either no fluid, 200 ml of cold (10°C) water, or 200 ml of warm (37°C) water every 20 min. In the hyperhydration / no rehydration trial, the final core (rectal) temperature was 0.2°C lower than during the euhydrated trial. In the hyperhydration / rehydration trial, the final core temperature was 0.6°C lower than the hyperhydration / no rehydration trial. These core temperature differences could be attributed to the hypothermic properties of the cold water ingested.

Gisolfi and Copping (35) calculated and reported that 1 L of 10°C water ingested, could have reduced body temperature by ~ 0.3°C to 0.4°C. The final core temperature difference was 0.8°C between the euhydration / no rehydration trial and the hyperhydration / rehydration trial, and they concluded that the hypothermic properties of water could not account for the observed difference. In both the hyperhydration and euhydration trials, the rehydration with warm water vs. cold water accounted for about a 0.2°C difference in T_{re}. Sweating rates were nearly identical during the final 90 min of

exercise in all the trials, but their discussion implies that sweating rate was greater in the first hour of exercise in the hyperhydration condition (but data was not provided to support this claim). They concluded that hyperhydration before exercise was not as effective in preventing hyperthermia as consuming an equal volume of warm or cold water while running, but also that combining hyperhydration before exercise with rehydration during exercise was the most effective hydration procedure for preventing hyperthermia.

Nadel et al. (72) studied the influence of hydration on circulatory and thermal regulation during 30-min cycle exercise (55%$\dot{V}O_2$max) in 35°C climate. Four subjects unacclimated to the heat performed three trials: 1) euhydration, 2) 2.7% hypohydration via diuretics, and 3) hyperhydration via ADH administration and water. They administered ADH by either nasal spray or subcutaneous injection and 10 min later gave the subjects 2 L of water. They reported the amount of ADH given reduced urine flow by ~ 30% of control level for 3-6 h. The subjects were unable to attain a steady-state core (esophageal) temperature in the 30-min exercise period in each experiment. The final core temperatures for the euhydration and hyperhydration experiments were nearly identical. Heart rate was lower during the hyperhydration than during the euhydration trials, but they did not observe any difference in either cardiac output or forearm blood flow.

Grucza et al. (42) studied the influence of hyperhydration on thermoregulation during 45 min of cycle exercise (52%$\dot{V}O_2$max) in a temperate (23°C) climate. Eight untrained subjects performed two exercise trials, the first when euhydrated and the second when hyperhydrated. Hyperhydration was attained by ingesting water (35 ml·kg $^{-1}$, 37°C) during the one hour preceding exercise. In the euhydration (control) trial, subjects exercised without any change in their normal water intake. The final exercise core (rectal) temperature was lower for the hyperhydration than euhydration trials: 37.7°C and 38.1°C, respectively. The sweating onset time and total body sweat loss was less during hyperhydration trial. The euhydration control trial was always tested first, so their results may reflect a trial order affect. They did not report any fluid consumption during exercise, and the lower sweating rates and higher core temperatures in the control trial suggest the control group was dehydrated.

Candas and colleagues (17) studied hydration effects on thermoregulatory and cardiovascular responses to prolonged (4 h) intermittent cycle exercise (70 W) in the heat (36°C). Five subjects unacclimated to the heat performed seven trials using the same exercise protocol: during the first two hours the work-rest cycle was 25 min exercise and 5 min rest, and during the second two hours the work-rest cycle was 20 min and 10 min, respectively. Three pre-exercise hydration regiments were used: euhydration, hypohydration and hyperhydration. For the hyperhydration trial, subjects were given 500 ml of isotonic electrolyte-sucrose solution (36°C) 30 min before exercise. Hyperhydration and hypohydration trials were evaluated with (rehydration) and without fluid replacement (dehydration) during exercise. The rehydration fluid was either an isotonic glucose-electrolyte beverage or water. The replacement fluid was given every 10 min to replace 80% of fluid lost during the euhydration trial. The euhydration-control trial was only evaluated without fluid replacement during exercise, so the comparison of hyperhydration to the control, again, would reflect the effects of hypohydration. They reported that hyperhydration had no effect on sweating rate. The core (rectal) temperature increase was less in the hyperhydration / rehydration trial; however, if rehydration was not employed, the rise in core temperature was nearly identical to the control trial. Also, the heart rate increase during exercise was lowest in the hyperhydration / rehydration trial, but this difference was eliminated when rehydration was not employed. For the purpose of evaluating the effects of hyperhydration, another control experiment should have been employed in which euhydration was maintained. However, this study does show that if hydration is not maintained, the magnitude of the core temperature increase will be similar to the control experiment.

Lyons et al. (61) studied the effects of glycerol mediated hyperhydration on thermoregulatory responses to exercise-heat stress. Six untrained subjects completed three trials in which they walked on a treadmill at 60% $\dot{V}O_2$ max for 1.5 h in a hot (42°C, 25% rh) climate. In one trial, they limited the fluid ingestion (L) to 5.4 ml·kg body weight $^{-1}$, and in the other two trials the subjects ingested a large volume of water (21.4 ml·kg $^{-1}$) in one hour with (G) or without (W) a bolus of glycerol (1 g·kg body weight^{-1}). Ninety minutes after this hyperhydration period, subjects began exercise. During exercise ~50 ml of fluid was ingested each hour. Prior to exercise, the water retained from hyperhydration was 80% in the glycerol trial as compared to 50% in the water trial. However, after exercise none of the hyperhydration fluid was retained in either trial. The

percent hypohydration levels at the end of trials were 0%, 0.3% and 0.7% for trials G, W and L, respectively; these hypohydration levels were calculated from their pre-drink body weights. Glycerol hyperhydration attenuated core (rectal) temperature increase by 0.7 °C and increased sweating rate by 3.6 $g \cdot min^{-1}$ compared to the water hyperhydration. The limited fluid trial was not a good control in determining the effects of hyperhydration on thermoregulatory responses because of the possible occurrence of hypohydration.

Montner et al. (68) studied the effects of water and glycerol hyperhydration on thermoregulation and exercise performance in a temperate climate. Eleven trained cyclist performed two exercise trials (75% $\dot{V}O_2$ max) to exhaustion in a temperate climate (~23 °C). In one trial (control) subjects drank 1.7 L of water, and in the other trial the subjects drank 1.7 L of glycerol/water solutions one hour before exercise. Hydration was not maintained during exercise in these trials. Prior to exercise the glycerol hyperhydration total body water was ~0.7 L greater than water hyperhydration trial. The investigators reported greater endurance times in the glycerol hyperhydration than water hyperhydration trials, 94 min and 77 min, respectively; however, they reported no differences in core (rectal) temperatures or sweating rates between trials. These investigators then repeated the two trials using seven subjects, but in this set of trials they included a 5% dextrose fluid replacement (0.6 $L \cdot h^{-1}$) drink during exercise. They reported similar total body water before exercise in the hyperhydration trials, but the endurance time was greater in the glycerol hyperhydration than water hyperhydration trial. They reported no difference in core temperature or sweating rate between hyperhydration trials, but did observe lower heart rates (3-6 $beats \cdot min^{-1}$) and greater endurance times during glycerol than water hyperhydration trials. They attributed the lower heart rate in the glycerol hyperhydration trial (compared to water hyperhydration trial) to an increased cardiac filling, but were unclear what mechanisms were responsible for increased endurance time.

Many variables can influence the results of studies on the effects of hyperhydration on thermoregulation, and these variables need to be controlled in order to evaluate the efficacy of hyperhydration. For example, the water temperature in the study by Moroff and Bass (69) was cool, and the resting core temperatures in the hyperhydration group were lower than the control group before exercise. The climate conditions, the exercise intensity and duration, and even the subjects state of heat

acclimation could all potentially influence the effects of hyperhydration on thermoregulation and exercise performance. For instance, subjects unacclimated to heat will have a much greater variability in their responses during exercise-heat stress for heart rate, core temperature and sweating rate. Also, with repeated exercise-heat exposures, subjects will become more heat acclimated; even though full acclimation often takes ~10 days, most of these adaptations occur in the first two to five exposures (91,105). Another very important variable is the state of hydration in the control trial during exercise. The control trial must maintain euhydration throughout the exercise in order for the effects of hyperhydration on thermoregulation to be evaluated correctly. Therefore, these confounding factors need to be properly controlled in order to evaluate the efficacy of hyperhydration during exercise.

THERMOREGULATION - UNCOMPENSABLE HEAT LOSS

Uncompensable heat stress is a condition in which the required evaporative cooling (E_{req}) is greater than the maximal capacity of the climate for evaporative heat loss (E_{max}) and during uncompensable exercise-heat stress body temperature will continue to rise during exercise. Ambient temperature, dew point, wind velocity, metabolic rate and clothing are important factors influencing whether exercise-heat stress will be a compensable or uncompensable condition. Uncompensable heat stress can be associated with heat exhaustion occurring at relatively low core temperatures (67,106). Exhaustion at low core temperature is thought to occur because the displacement of blood to the skin (via increased cutaneous dilation and compliance) causes marked cardiovascular strain and instability (89,94). During uncompensable heat stress conditions, a hyperhydration method, which increases total body water and may defend blood volume for extended periods, should theoretically decrease cardiovascular strain and improve exercise-heat performance regardless of any thermoregulatory effects. Hypohydration has been shown to decrease physiological tolerance to exercise-heat strain during uncompensable heat stress (106). The ergogenic effects of hyperhydration have not been evaluated during uncompensable heat stress conditions.

Sawka et al. (106) studied the effects of hypohydration on physiologic tolerance during uncompensable exercise-heat stress. The observed that exhaustion rates at a given level of physiological strain were greater when hypohydrated than euhydrated during exercise in an uncompensable heat stress environment. Seventeen heat acclimated men performed two exercise-heat stress tests (45% $\dot{V}O_2$max, 49°C, 20%rh): one euhydrated and one hypohydrated (8% of total body water, TBW). They reported that hypohydration reduced the core (rectal) temperature that could be tolerated, and final heart rate was greater when compared to the euhydration trial. Hypohydration was thought to reduce physiologic tolerance to heat strain because of the decreased blood volume resulting in less cardiac filling.

Montain et al. (67) studied the influence of exercise intensity, protective clothing and climate on physiological tolerance to uncompensable heat stress. Seven heat acclimated men attempted 180-min treadmill exercise (425 and 600 W) heat stress tests wearing full or partial protective clothing. They observed that being fully clothed in protective clothing reduces physiological tolerance, as core (rectal) temperature at exhaustion was lower in full than in partial clothing. They also reported the exercise intensity and climate did not alter physiological tolerance to uncompensable heat stress. Wearing protective clothing was thought to reduce physiologic tolerance to heat strain because of the higher skin temperatures causing greater displacement of blood from the central circulation to the skin resulting in less cardiac filling.

Nielsen et al. (74) studied the effects of heat acclimation in an uncompensable heat stress condition. They had eight highly trained subjects cycle to exhaustion at 60% of their $\dot{V}O_2$max for nine to twelve days in a 40°C climate. They reported the final core (esophageal) temperature was consistently 39.7°C at exhaustion and was not changed with heat acclimation, however, endurance time was increased. They also observed increased sweating rate, increased cardiac output, and reduced heart rate at exhaustion after heat acclimation. The state of heat acclimation did not appear to affect physiologic tolerance to heat strain during uncompensable exercise-heat stress in these trained subjects. They attributed the improved performance (heart rate, stroke volume and cardiac output) to a 13% increase in plasma volume.

Together these studies indicate that during uncompensable heat stress adjustments in clothing (67) and hydration (106) affected physiological tolerance, but

aerobic fitness (106), exercise intensity (67), heat acclimation (74) and climate (67) did not. The effect of hyperhydration on physiologic tolerance to heat strain has not been evaluated during uncompensabe exercise-heat stress. It is anticipated that hyperhydration will increase blood volume (30), and allow greater cardiac filling, and therefore, better able to maintain cardiac output during uncompensable exercise-heat stress.

MILITARY APPLICATION

Military missions conducted in hot environments (e.g., Desert Shield Operation/Storm, Haiti, Somalia) often result in soldiers working long hours without adequate fluid replacement. This leads to performance decrements and increased incidence of heat casualties (46). Soldiers wearing nuclear-biological-chemical (NBC) protective clothing, self-contained toxic environment protective outfit (STEPO) or other devices that impede the soldier's ability to obtain or drink fluids are likely to dehydrate and become hypohydrated while working in these ensembles (5,16,116). A recent study (21) has shown that 80% of the soldiers wearing NBC protective clothing were dehydrated and about 15% of these exceeded a 5% reduction in body weight after 12 h of maneuvers. The low moisture permeability and high insulating properties of NBC protective clothing prevents normal dissipation of body heat and results in a greater body heat storage, higher core temperature, greater sweating rate, and greater rate of dehydration. Subsequently, this cascade of events will result in decreased performance and an increased probability of heat injury.

If hyperhydration has any thermoregulatory or performance advantages, then there are many situations (military, industrial, agricultural, sports, and recreational) that hyperhydration would directly benefit. If the state of hyperhydration could be achieved and maintained prior to a heat stress situation, the adverse effects of hypohydration may be delayed, decreased or even eliminated depending on the severity of the heat stress. We do not know if hyperhydration will improve physiological tolerance, improve performance or delay exhaustion during uncompensable heat stress. Increasing total body water of soldiers by hyperhydration using an aqueous glycerol solution could be beneficial during exercise-heat exposure, especially for missions in which the soldiers are prone to dehydration or in an uncompensable heat stress environment.

Three studies (30,61,81) suggest that hyperhydration with glycerol increases body water, and one study (61) has shown that heat strain during exercise is markedly reduced compared to the control trial. Studies (61,69,75) that have reported lower core temperatures or greater sweating rates have had inadequate control trials in which they did not replace fluid lost during exercise; therefore, the differences reported may not be due to the effects of hyperhydration but may be due to the effects of hypohydration in the control group. Maintaining euhydration during exercise is essential to determine the efficacy of hyperhydration on thermoregulation during exercise-heat stress. No study has evaluated the effects of hyperhydration during uncompensable exercise-heat stress. This study evaluated whether water hyperhydration or glycerol-hyperhydration would improve thermoregulatory responses during compensable exercise-heat stress and improve soldier performance during uncompensable exercise-heat stress.

MATERIALS AND METHODS

This study consisted of two phases: Phase I evaluated the thermoregulatory effects of hyperhydration during compensable exercise-heat stress and consisted of five trials. Phase II evaluated the effects of hyperhydration on cardiovascular responses and physiological tolerance to heat strain during uncompensable exercise-heat stress and consisted of three trials.

SUBJECTS

Eight healthy male soldiers stationed at Solders System Command, Natick, MA, completed all the trials in Phase I and Phase II. Prior to exercise-heat stress tests (HSTs) the subjects were heat acclimated. All subjects received a physical examination including a medical history for medical clearance prior to any testing.

Subjects were fully informed of all aspects of the study and signed a statement of informed consent approved by the Human Use and Review Committee (HURC), U.S. Army Research Institute of Environmental Medicine, Natick, MA and by the Human Subjects Research Review Board, Office of the Surgeon General, Department of the

Army, Falls Church, VA. This study adhered to AR 70-25 and United States Army Medical Research and Materiel Command Regulation 70-25 on Use of Volunteers in Research. Hazards and risks associated with procedures performed in this study were adhered to as described in the U.S. Army Research Institute of Environmental Medicine (USARIEM), Type Protocol, "Human Research Studies in the Areas of Thermal, Hypoxic, and Operational Stress, Exercise, Nutrition and Military Performance," dated December 1995.

STUDY DESIGN

The schedule for administration of procedures and tests is shown in **Table 2**. Preliminary measurements were made before any exercise-heat stress tests (HSTs). These measurements included $\dot{V}O_2$max, submaximal workload determination, body composition measurements, and total body water (TBW). The HSTs were administered in random order for each phase of this study; however, Phase II trials always followed the completion of Phase I trials. All HSTs were scheduled on nonconsecutive days.

Table 2. Study Schedule

Week 1	$\dot{V}O_{2max}$ Tests Submax $\dot{V}O_2$ Measurements Underwater Weighing
Weeks 1-3	Heat Acclimation Total Body Water Measurements
Weeks 3-7	Exercise-Heat Tests (HSTs)
Weeks 7-8	Repeat $\dot{V}O_{2max}$ Tests For Volunteers Testing in Phase II

All tests were conducted at the U.S. Army Research Institute of Environmental Medicine, Natick, MA from February through May, 1995. During all experiments, the subjects wore shorts, tennis shoes and socks unless otherwise specified. In addition, nude body weights were measured for two weeks in the morning after voiding before breakfast. These body weights were used to establish baseline body weights which represent euhydration. Percent body fat, maximal oxygen uptake ($\dot{V}O_2$max) and total body water were measured in the first 3 weeks. The HST's submaximal exercise

intensity was determined in a temperate climate (22 to 24°C db; ~25 -29% rh). Submaximal exercise intensity (~ 45%) and treadmill settings were determined prior to any HSTs and ranged from 0% to 8% grade and from 0.89 to 3.12 $m \cdot s^{-1}$ velocity. Oxygen uptake was measured while subjects walked on the treadmill for 5 to 10 min periods at different grades and velocities. Prior to HSTs subjects practiced 1) walking at the selected treadmill grade and speed (that corresponded to ~45% of their $\dot{V}O_2$ max), 2) cardiac output procedures (CO_2 rebreathing), 3) placement and wearing the esophageal probe and 4) wearing of rectal probes in order to become familiar with techniques and procedures used during the HSTs.

TEST SESSIONS AND PROCEDURES

The subjects body density was measured by hydrostatic weighing and residual lung volume was measured while submerged underwater. Percent body fat and lean body mass were calculated from body density by using the equation of Siri (111). Maximal $\dot{V}O_2$ was determined from a progressive intensity and continuous effort treadmill protocol (32). The initial treadmill grade was set at zero, and increased 2.5% grade every 1.5 min. Treadmill velocity (2.68 or 3.13 $m \cdot s^{-1}$) was determined from the heart rate response at the end of a 10-min warm-up walk (1.56 $m \cdot s^{-1}$ with a 10% grade). If heart rate was greater than 145 $beats \cdot min^{-1}$, the velocity was set at 2.68 $m \cdot s^{-1}$; if heart rate was less than 145 $beats \cdot min^{-1}$, velocity was set at 3.13 $m \cdot s^{-1}$ for the $\dot{V}O_2$ max test (52). Established criteria were employed for determination of maximal $\dot{V}O_2$ values (63,117). Maximal values were determined when final treadmill grade could not be tolerated and when the difference in $\dot{V}O_2$ values between final exercise stages was less than 1.5 $ml \cdot kg^{-1}$ for 2.68 $m \cdot s^{-1}$ treadmill velocity or less than 2.1 $ml \cdot kg^{-1}$ for the 3.13 $m \cdot s^{-1}$ velocity.

Subjects were heat acclimated by walking at ~45% $\dot{V}O_2$max on a treadmill for two 50-min bouts spaced by a 10-min rest period for six to ten days in a hot-dry climate (T_a= 35°C, rh = 45%, air velocity = 1 $m \cdot s^{-1}$). Heat acclimation was determined when non-significant differences were observed in final exercise core temperatures and heart rates on two continuous days of acclimation. During rest and exercise, the subjects were encouraged to drink. Heart rate and rectal temperature were measured during the heat acclimation sessions. Subjects discontinued exercise if rectal temperature

reached 39.5°C, or if heart rate achieved 90% of maximum for a 5-min period. Body weights (nude and clothed) were obtained before and after exercise. Subjects were asked to participate in additional heat acclimation sessions on non-test days if they had greater than two consecutive days without an exercise-heat exposure.

Total body water was measured using the deuterium-labeled water dilution technique (31,108) in the final week of acclimation. This measurement was performed the morning following eight hours of abstinence from food and drink, and with the subjects seated. Subjects first provided a 10 ml blood sample, and then drank 30 grams of deuterium oxide (D_2O) followed by two 30 ml rinses of tap water. A serum blood sample was collected 3 h after D_2O ingestion, and deuterium was measured in the sample. The serum sample (2 ml) was diluted in an equal volume of deionized water, and the D_2O was allowed to equilibrate for 48 h at 37°C in Conway diffusion dish (Bel-Air Products, Pequannocol, NJ) sealed with Parafilm. Each sample was purified in triplicate, and absorption was measured in a calcium fluoride cell seated in a water-cooled jacket. Samples were allowed to equilibrate in the cell at 15°C for 6 min prior to measurements using an infrared spectrophotometer with a fixed filter (Miran 1FF, Foxboro, MA). D_2O concentrations were calculated from a standard curve, (r=0.99) and TBW was calculated.

HYDRATION PROCEDURES

Hyperhydration began immediately after venous catheterization/venepuncture and plasma osmolality measurement. In the hyperhydration trials, subjects first drank 3.9 ml·kg lean body mass $(LBM)^{-1}$ of the experimental solution (i.e., either glycerol or water solution sweetened with aspartame). The experimental solution administration was double-blind; the water and glycerol solutions were of similar sweetness (Aspartame), color, flavor and temperature (10°C) in order to mask the taste of glycerol. The glycerol solution contained 1.2 g glycerol·kg LBM^{-1} and was of purity for human consumption. Following ingestion of the experimental solution, the subject drank a large volume (25.2 ml·kg LBM^{-1}) of water (36°C). The total volume of fluid consumed in a 30-min period was 29.1 ml·kg LBM^{-1}. This hyperhydration method is identical to that reported by Freund et al. (30).

Fluid replacement during exercise in trials Euhydration (Eu), Glycerol-Rehydration (GR) and Water-Rehydration (WR, see **Table 3**) was an attempt to replace fluid loss during exercise as measured on the last day of heat acclimation. The rehydration fluid was water (~36°C) given in equal volumes at approximately 20, 40, 60, 80 and 100 min of exercise.

EXERCISE-HEAT STRESS TESTS, PHASE I

Phase I consisted of five HSTs: euhydration (Eu), glycerol hyperhydration with no replacement (GD), glycerol hyperhydration/ rehydration (GR), water hyperhydration with no replacement (WD) and water hyperhydration/ rehydration (WR) as shown in **Table 3**. The day before the HSTs, each subject was instructed not to eat for 8 h before the report time; he was weighed and instructed to drink 2 L of a provided commercial carbohydrate electrolyte beverage by 2200 h the evening before the HST. The next morning the subject reported to the laboratory, and a nude body weight was taken after voiding. Subjects wore shorts, socks, and running shoes during the HST.

A flexible Teflon catheter was inserted in a superficial arm vein and plasma osmolality was measured to ascertain euhydration status. The subjects began drinking the hyperhydration solution 1 h prior to exercise-HST which they had to complete within 30 min. The subject swallowed an esophageal probe and inserted the rectal probe for core temperature measurements. Subjects entered the climatic chamber 30 min prior to exercise and were instrumented (temperature probes, ECG leads). Clothed body weights were measured before exercise. Body temperatures and heart rates were continuously monitored and metabolic rate was measured at ~5, ~55 and ~100 min of exercise. After the exercise, subject's body weights (clothed and nude) were again measured.

Venous blood samples (10 ml) were taken after standing for ~25 min in the climatic chamber prior to exercise, and three samples were taken during exercise (~40 min, 80 min and a final exercise sample).

Table 3. Phase I: Compensable Heat Stress Trials

Trial	Pre-Exercise Hydration	Exercise-Fluid Replacement
Eu	Euhydrated	With fluid replacement
GD	Hyperhydration (glycerol)	No fluid replacement
GR	Hyperhydration (glycerol)	With fluid replacement
WD	Hyperhydration (water)	No fluid replacement
WR	Hyperhydration (water)	With fluid replacement

Exercise HSTs consisted of subjects attempting 120 min of treadmill exercise (1.56 to 1.65 $m \cdot s^{-1}$ at 4% to 9% grade = 45% of $\dot{V}O_2$max) in the heat (T_a= 34.9±0.1°C, T_{dp} = 25.9±0.6°C, air velocity = 1 $m \cdot s^{-1}$). For each subject, HSTs were conducted about the same time of day. The exercise-heat stress at ~45% $\dot{V}O_2$max in a 35°C, 45% rh climate requires essentially all heat loss via evaporation. The required evaporative heat loss (E_{req}) was less than the maximal capacity of the climate for evaporative heat loss (E_{max}) and, therefore, these trials were considered compensable exercise-heat stress tests. For these conditions (climate, clothing and exercise), the United States Army Research Institute of Environmental Medicine's Heat Strain Prediction Model (77) predicted a compensable heat stress condition in which subjects would be able to complete 120-min HSTs before reaching physiologic criteria.

PHYSIOLOGICAL MEASUREMENTS, PHASE I

Skin temperatures were measured using a five point (forearm, upper-arm, chest, thigh and calf) thermocouple skin harness, and mean skin temperature ($\bar{T}_{sk}$) was calculated (80). Esophageal temperature (T_{es}) was measured from a thermocouple placed at the level of the heart (distance from the naris to the heart is approximately

0.25 x height) and it was adjusted ±2 cm to where the highest temperature was recorded (104). The probe was marked and taped in place to prevent it from moving. Since saliva lowers T_{es} measurements, the subject was asked to avoid swallowing by spitting saliva into a cup during HSTs. The rectal temperature (T_{re}) was measured from a thermistor inserted 10 cm beyond the anal sphincter (104). Body temperatures were continuously measured during exercise. Local sweat rate ($\dot{m}_{sw}$) of the upper arm was measured by automated dew-point sensors enclosed in a ventilated capsule (40). Room dew point was also measured near the subject using the same type of sensor. Total body sweating rate was calculated from pre- and post-exercise weights and was corrected for water intake and urine output.

Metabolic rates were measured at approximately 5, 55 and 100 min of exercise. Oxygen uptake and carbon dioxide production were measured by open circuit spirometry using the automated system (Sensormedics 2900). The automated system was calibrated using standard gas mixtures and verified after each data collection period. Heart rate was continuously monitored and recorded every 10 min from a CM5 electrode placement.

Venous blood samples were collected from an indwelling Teflon catheter placed within a superficial arm vein. Patency was maintained with heparinized saline; the catheter was flushed with ~2 ml of blood before each 8 ml sample was obtained. A blood sample was taken at rest while the subject stood in the climatic chamber, and three exercise blood samples were obtained while the subject walked on the treadmill. A Blood sample was drawn into appropriate anticoagulant-containing syringes and placed on ice. Blood variables measured in triplicate include hemoglobin (hgb), hematocrit (hct), lactate, sodium, potassium, osmolality, glycerol and protein. Percent change in plasma volume and blood volume were calculated from the appropriate hemoglobin and hematocrit values. Microhematocrit (Hct) was determined by centrifugation while Hgb concentration was determined by hemoglobinometer (Coulter Electronics). Serum was analyzed for sodium (Na^+) and potassium (K^+) by ion specific electrode (IL) and for osmolality by freezing point depression (Advanced Micro-Osmometer, Advanced Instruments, Model 3MO). Plasma protein concentration was determined by refractory photometer (Schuco Model 5711-2020). Serum glycerol levels were determined using commercial test kits (triglyceride kit for free glycerol, Sigma Diagnostics Inc.) for application on an IL Monarch. Urine volumes were measured after

fluid consumption, before exercise, during the HSTs and immediately following the exercise session.

EXERCISE-HEAT STRESS TRIALS, PHASE II

Phase II HSTs were performed with subjects wearing chemical protective clothing (water vapor permeability index (i_m) ~0.33 and clo~2.0). Subjects performed three HSTs (see **Table 4**): euhydration with no rehydration (CON), GD and WD. They entered the environmental chamber 30 min after beginning hyperhydration and 30 min before exercise. After instrumentation, the subject dressed in a chemical protective suit; Battle Dress Uniform (BDU), Battle Dress Overgarment (BDO), boots, mask, and hood. The subjects exercised at the same speed and treadmill grade as in the compensable heat stress trials. The required evaporative heat loss (E_{req}) exceeded the maximal capacity of the climate for evaporative heat loss (E_{max}), therefore, these trials were uncompensable exercise-heat stress tests. The U.S.A. Research Institute of Environmental Medicine's Heat Strain Prediction Model indicated the subjects would tolerate ~ 40 min of exercise in this environment (77).

Rectal temperature, skin temperature and heart rate were continuously monitored as described. Metabolic and cardiac output measurements were made at 10, and 20 min. Blood pressure was measured at rest, 10, 20 and 30 min of exercise. A blood sample was obtained by venepuncture from an antecubital vein before hyperhydration and was analyzed for an initial plasma osmolality.

Table 4. Phase II: Uncompensable Heat Stress Trials

Trial	Pre-Exercise Hydration	Exercise Fluid Replacement
CON	Euhydration	No fluid replacement
GD	Hyperhydration (glycerol)	No fluid replacement
WD	Hyperhydration (water)	No fluid replacement

PHYSIOLOGICAL MEASUREMENTS, PHASE II

Skin temperatures and rectal temperatures were measured as described in Phase I. Total body sweating rates were calculated from pre- and post-exercise weights and corrected for water intake and urine output.

Metabolic rate was measured at 10 and 20 min of HST. Oxygen uptake and carbon dioxide (CO_2) production were measured by open circuit spirometry using an automated system (Sensormedics 2900). Cardiac output was measured using CO_2 equilibration method (45). This method required the subject to rebreathe a 10% to 12% CO_2 gas concentration with balance gas mixture of O_2 from a 5 L bag with a volume 1.5 times his tidal volume. After a normal expiration, the subject was switched to the bag and instructed to breathe deeply at a rate of 30 breaths·min^{-1} until CO_2 equilibrated between the bag and lungs. The partial pressure of venous CO_2 ($PvCO_2$) was estimated from the PCO_2 plateau which occurred when alveolar and bag PCO_2 were equal to mixed venous blood PCO_2. The partial pressure of end tidal CO_2 ($PETCO_2$) was used to predict PCO_2 of arterial blood ($PaCO_2$) using Jone's (51) equation. Blood CO_2 contents were derived using a standard CO_2 dissociation curve and the Fick equation was used to calculate cardiac output. Heart rate was continuously monitored and recorded every 10 min from a CM5 electrode placement. Blood pressure was measured using an automated system, Suntech 4240, at rest, 10, 20 and 30 min of exercise. Urine volume was measured following each urine collection.

CALCULATIONS AND STATISTICS

Calculations

The percent changes in plasma volume (%ΔPV) was calculated from the Hct and Hgb values using the following equation (20).

$$\%\Delta PV = 100 \times \frac{Hgb_{pre}}{Hgb_{post}} \times \frac{1-Hct_{post} x10^{-2}}{1-Hct_{pre} x10^{-2}} - 100$$

Hgb = hemoglobin
Hct = hematocrit

The erythrocyte volume (RCV) and blood volume (BV) was calculated using the following equations (107):

$$RCV = 0.028\ (LBM) + 0.185$$
$$BV = 0.072\ (LBM) + 0.584$$

LBM = Lean Body Mass

Heat exchange was calculated using the heat balance equation:

$$S = M \pm W \pm E \pm R \pm C \pm K$$

S = heat storage
M = metabolic heat production
W = work
E = evaporative heat exchange
R = radiation
C = convection
K = conduction.

The required evaporative cooling at the skin (E_{req}) was calculated with the following formula from Gagge and Nishi (34) and modified by Kraning (54):

$$E_{req} = M_{net} + (R + C)\ (\text{in } W \cdot m^{-2})$$

M_{net} = metabolic rate in $W \cdot m^{-2}$
R +C = sum of radiative and convective heat exchange
= sum of linear radiative heat transfer coefficient (4.7 $W \cdot m^{-2} \cdot {}^{\circ}C^{-1}$) and convective heat transfer coefficient (7.8 $W \cdot m^{-2} \cdot {}^{\circ}C^{-1}$) multiplied by the gradient between skin temperature ($\bar{T}_{sk}$) and ambient temperature (T_a) (95)

The maximum evaporative heat exchange was estimated using the following equation (38):

$$E_{max} = 2.2 \cdot 6.46 \left[i_m / _{clo} \right] \cdot A_D \cdot (P_{s,sk} - P_a)$$

$i_m/_{clo}$ = clothing value for specific velocity ($m \cdot s^{-1}$) of air movement
A_D = Dubois total surface area of the body (m^2)
$P_{s,sk}$ = saturated vapor pressure of skin temperature ($\bar{T}_{sk}$) (Torr)
(estimated using Antoine's equation (95))
P_a = ambient water vapor pressure (Torr)

2.2 = Lewis relation (°C/Torr)
6.46 = conductance conversion of clo to W

The body surface area was calculated using the DuBois formula (22):

$$A_D = W^{0.424} \cdot H^{0.725} \cdot 71.84$$

W = body mass (Kg)
H = height (cm)

Local sweating rate was calculated:

$$\dot{m}_{sw} = \Delta P_{H2O} \cdot (AF) / (R_w \cdot A \cdot T)$$

ΔP_{H2O} = water vapor pressure gradient between ambient air and skin
AF = air flow through capsule ($L \cdot min^{-1}$)
R_w = gas constant for water vapor 3.464 ($Torr \cdot l \cdot g^{-1} \cdot {}^oK^{-1}$)
A = area of sweat capsule (15.9 cm^2)
T = absolute temperature (°K) of dew point

Sweating sensitivity was the slope of the regression line when $\dot{m}_{sw}$ was plotted as function of T_{es} during the first 20 min of exercise. The threshold for local sweating was the T_{es} when $\dot{m}_{sw}$ exceeded .06 $mg \cdot cm^{-2} \cdot min^{-1}$ (66).

Mean skin temperature ($\bar{T}_{sk}$) was calculated using the appropriate weighing (80) by the following equation:

$$\bar{T}_{sk} = 0.3[t_{chest} + t_{arm}] + 0.2[t_{thigh} + t_{leg}]$$

t_{chest} = skin temperature of mid-pectoralis surface (°C)
t_{arm} = skin temperature of mid-forearm (ventral surface)(°C)
t_{thigh} = skin temperature of mid-thigh surface (°C)
t_{leg} = skin temperature of mid-gastrocnemius surface (°C)

Mean body temperature ($\bar{T}_b$) was calculated by (33)

$$\bar{T}_b = 0.9 (T_{re}) + 0.1(\bar{T}_{sk})$$

T_{re} = rectal temperature (°C)
$\bar{T}_{sk}$ = mean skin temperature (°C)

Cardiac output (Q) was calculated using the Fick equation (50):

$$Q = \frac{\dot{V}CO_2}{CvCO_2 - CaCO_2}$$

$\dot{V}CO_2$ = volume of CO_2 produced
$CvCO_2$= mixed venous CO_2 content
$CaCO_2$= arterial CO_2 content

The partial pressure of CO_2 in arterial blood ($PaCO_2$) was calculated using the partial pressure of end tidal CO_2 ($PETCO_2$) and Jone's (51) equation:

$PaCO_2$= 5.5 + 0.90 $PETCO_2$ - 2.1V_T

V_T = Tidal Volume (L)

Mean arterial pressure (MAP) was calculated from the following equation (85):

MAP = Diastolic Pressure + $^1/_3$ [Systolic Pressure - Diastolic Pressure] (mmHg)

Total peripheral resistance (TPR) was calculated by (85):

TPR = MAP ÷ Cardiac Output (PRU)

Statistics

Descriptive analyses included calculation of means, standard deviations, standard errors, and Pearson product-moment correlations. The experimental design employed repeated measures testing. Analyses of variance with repeated measures was used to determine if hyperhydration had significant or interactive effects. Student-Newman-Keuls pairwise multiple comparison procedures were used to identify differences between means when statistical significance was achieved. Statistical significance was accepted with alpha level of $P<0.05$.

RESULTS

Data presented in the text are means ±SD and data presented in tables and figures are means ±SE unless otherwise indicated. **Table 5** presents the physical characteristics (age, body mass, lean body mass, body surface area, maximal oxygen uptake and total body water) of the eight male volunteers who participated in this study. Their average age was 23±6 years and ranged from 19 to 36 years. Body mass was 76±15 kg and ranged from 56 to 100 kg. Lean body mass was 63±9 kg and ranged from 53 to 73 kg. $\dot{V}O_{2\ max}$ was 56±8 $ml \cdot kg^{-1} \cdot min^{-1}$ and ranged from 42 to 69

Table 5. Subject Characteristics

Subject	Age (Yr)	Body Mass (kg)	LBM (kg)	BSA (m^2)	$\dot{V}O_{2\ max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	TBW (L)
1	19	76.2	66.7	1.98	61.2	45.4
2	20	63.1	55.2	1.67	59.1	38.4
3	19	62.9	52.3	1.73	54.7	40.0
4	21	74.3	67.5	1.88	51.0	51.6
5	19	82.6	66.2	2.06	59.8	51.7
6	26	100.6	72.7	2.16	42.2	50.0
7	26	91.2	73.1	2.12	53.7	54.5
8	36	56.1	52.4	1.63	68.9	39.4
Mean	23	75.9	63.3	1.90	56.3	46.4
SD	±6	±15.2	±8.7	±0.07	±7.9	±6.4

Abbreviations: LBM = Lean Body Mass, BSA = Body Surface Area,
$\dot{V}O_{2\ max}$ = Maximal oxygen uptake, SD = Standard Deviation

$ml \cdot kg^{-1} \cdot min^{-1}$. In addition, total body water was 46.4±6.4 L and ranged from 38 to 54 L. The required evaporative heat loss (E_{req}) needed to maintain thermal equilibrium was 293±17 $W \cdot m^{-2}$ and 366±20 $W \cdot m^{-2}$ for Phase I and Phase II trials, respectively. The average maximal capacity of the climate for evaporative heat loss (E_{max}) was 462±89 $W \cdot m^{-2}$ and 88±16 $W \cdot m^{-2}$ for Phase I and Phase II trials, respectively. Therefore the percentage of E_{req}/E_{max} was 63% and 415% for compensable and uncompensable exercise-heat stress, respectively.

PHASE I, COMPENSABLE EXERCISE-HEAT STRESS

Preliminary Hydration Measurements

Prior to each trial, pre-drink body masses were similar ($P > 0.05$) between trials. For Eu, GD, GR, WD and WR trials body masses were 76.5±4.6, 76.4±4.7, 76.1±14.6, 76.1±14.6, and 76.1±14.4 kg, respectively. Pre-drink plasma osmolalities were similar ($P > 0.05$) between trials. Plasma osmolalities for Eu, GD, GR, WD, and WR trials were 282±3, 283±5, 284±5, 284±5 and 284±5 mosmol/kg H_2O, respectively.

Fluid Ingestion

Eight subjects completed all trials in Phase I of this study. A ninth subject was unable to ingest the glycerol solution without becoming nauseous; this subject was removed from the study. Two of the eight subjects on one occasion vomited after drinking the glycerol solution so the trial was aborted and repeated on another day. One of the eight subjects was unable to complete the 120-min exercise period in two of the five trials. No trials were stopped because of temperature or heart rate criteria. For a given subject, the same amount of fluid was consumed before each hyperhydration trial, and the same rate of water consumption occurred during exercise in each rehydration trial. The volume of fluid consumed before each hyperhydration trial was 1.84±0.25 L. The volume of water consumed during exercise in each rehydration trial was 2.36±0.51, 2.23±0.72 and 2.19±0.52 L during Eu, GR and WR trials, respectively. These values were not different ($P>0.05$) and the between trial variability was due to differences in exercise duration or subject drinking tolerance.

Figure 1 presents change in total body water (TBW) during each trial. For euhydration trials, TBW did not change over time either before or during exercise. For hyperhydration trials GD, GR, WD, and WR, TBW increased ($P<0.05$) by 1.40±0.39, 1.38 ±0.33, 1.50 ±0.40 and 1.54 ±0.31 L, respectively at 30 min after drinking with no difference ($P>0.05$) between trials. For all hyperhydration trials, TBW decreased during exercise; however, the decreases were less ($P<0.05$) in GR and WR trials than GD and WD trials. There were no differences ($P>0.05$) in TBW between the GR and WR trials or between GD and WD trials during exercise. Total body water increased by 1.86% and 1.45% from before drinking to after exercise-heat exposure during GR and WR trials, respectively. This corresponded to retaining 21% and 17% of the consumed fluid for GR and WR trials, respectively. No differences ($P>0.05$) were found between GR and WR trials for either TBW increase or percentage of fluid retained.

Figure 2 presents the change in plasma volume for each trial. There were no differences in plasma volume between trials Eu, GD, GR, WD, and WR. There were significant ($P<0.05$) decreases in plasma volumes from pre-exercise (Ex0) to the final exercise (Ex120) value in both GD and GR trials. Plasma volume changes were calculated from hemoglobin and hematocrit data that are presented in **Table 6**.

Figure 3 presents total urinary output for each trial. Total urinary output values were greater ($P<0.05$) in hyperhydration (GD, GR, WD, and WR) than in Eu trials. The total urinary outputs for the trials Eu, GD, GR, WD, and WR were 0.15 ±0.18, 0.52 ±0.38, 0.61 ±0.19, 0.71 ±0.34 and 0.70 ±0.25 L, respectively. No differences ($P>0.05$) in total urinary output were observed between glycerol hyperhydration trials and water hyperhydration trials.

Figure 4 presents serum osmolality values during each trial. Pre-exercise (Ex0) serum osmolality was greater ($P<0.05$) in GD and GR than Eu trials. The mean total osmolar load from the ingested glycerol was 802 mOsmol or 16.6 mOsmol·L^{-1} TBW. Pre-exercise serum osmolality values were lower ($P<0.05$) during WD and WR than Eu trials. During exercise serum osmolality increased ($P<0.05$) in GD and WD trials, but did not change ($P>0.05$) during exercise in other trials.

FIGURE 1

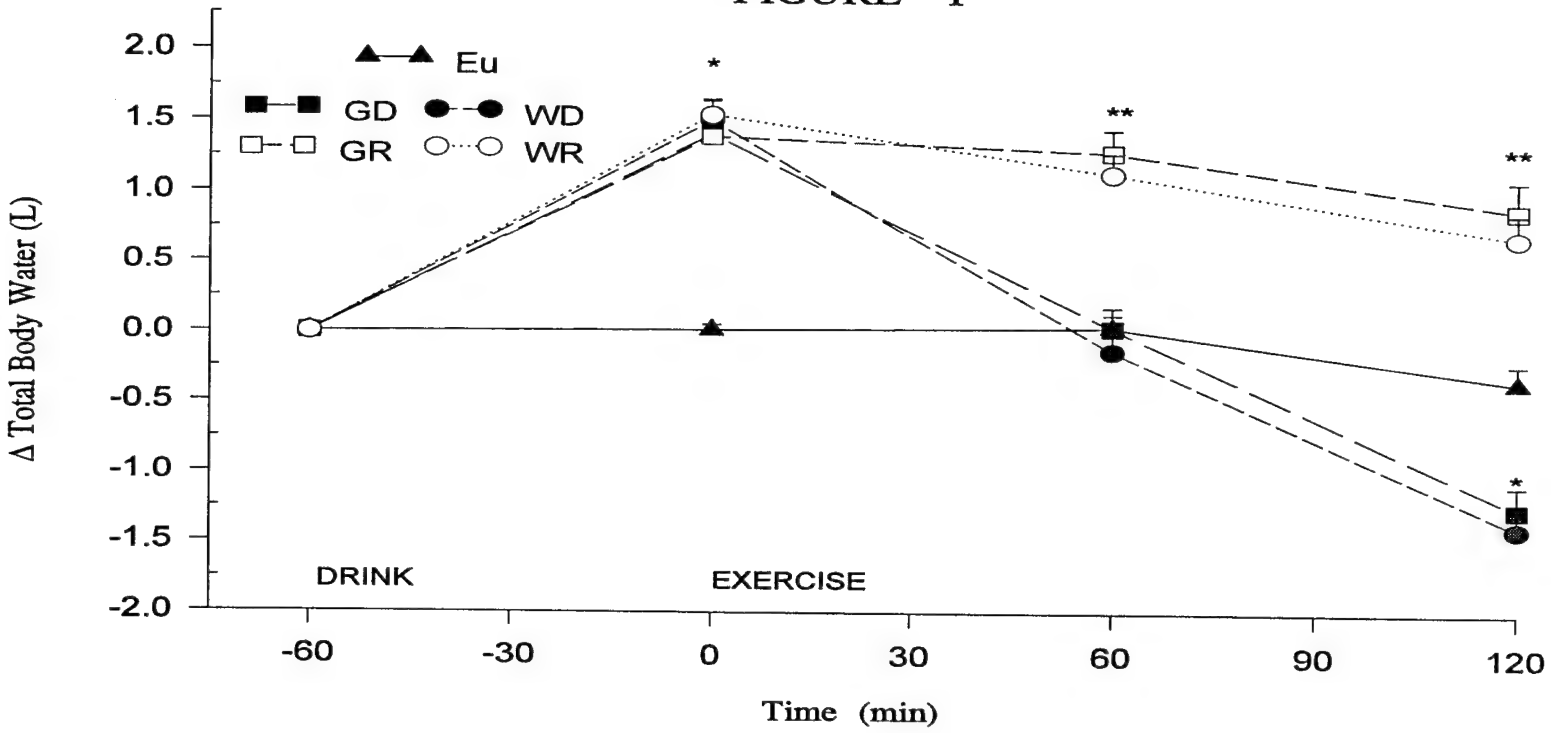

FIGURE 1: Changes in total body water over time during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration. *Significantly ($P<0.05$) different from Eu trial. **Significantly ($P<0.05$) different from GD and WD.

FIGURE 2

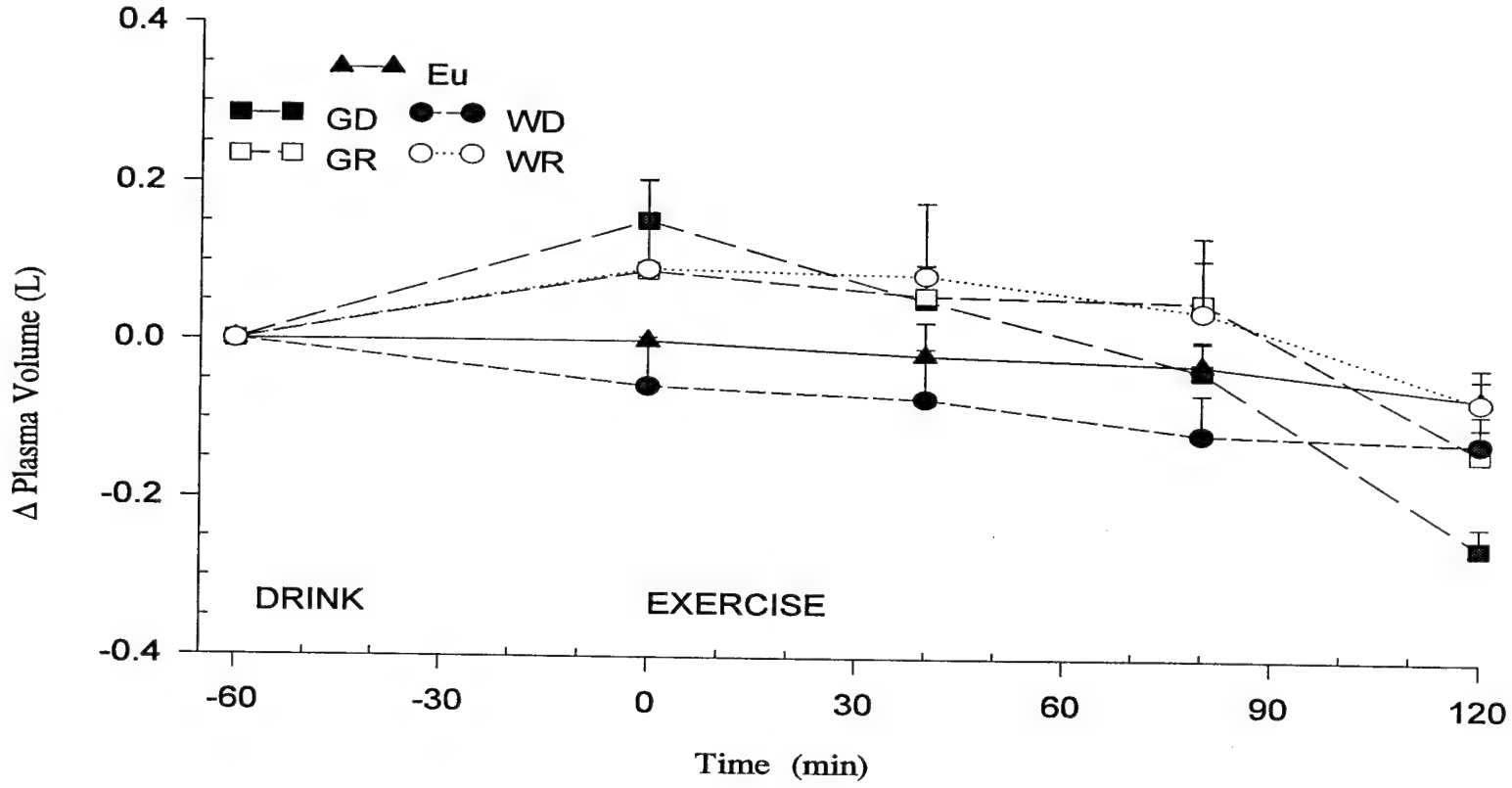

FIGURE 2: Changes in plasma volume over time during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration.

Table 6. Mean ±SE Hematologic Values Obtained Before and During HSTs

Trial	Time	Hemoglobin ($g \cdot dl^{-1}$)		Hematocrit (%)		Total Protein ($g \cdot dl^{-1}$)	
Eu	Ex0	15.7	±0.3	44.8	±1.0	7.0	±0.1
	Ex40	15.8	±0.4	45.0	±1.2	7.1	±0.2
	Ex80	15.7	±0.4	45.2	±1.2	7.1	±0.2
	Ex120	15.9	±0.4	45.5	±1.2	7.3	±0.2
GD	Ex0	15.2	±0.4	43.8	±0.7	6.7	±0.2
	Ex40	15.5	±0.4	44.4	±1.0	6.9	±0.2
	Ex80	15.9	±0.3	44.8	±0.9	7.1	±0.2
	Ex120	16.2	±0.4	45.2	±0.8	7.4	±0.2
GR	Ex0	15.3	±0.3	44.5	±1.0	6.8	±0.2
	Ex40	15.4	±0.3	44.5	±0.9	6.8	±0.2
	Ex80	15.4	±0.3	44.8	±1.0	6.9	±0.2
	Ex120	15.6	±0.3	45.3	±1.1	7.1	±0.2
WD	Ex0	15.6	±0.4	46.4	±1.2	6.9	±0.2
	Ex40	15.8	±0.4	46.1	±1.1	6.9	±0.2
	Ex80	16.1	±0.4	45.9	±1.1	7.2	±0.2
	Ex120	16.4	±0.4	46.4	±1.1	7.5	±0.2
WR	Ex0	15.3	±0.4	44.6	±1.4	7.0	±0.2
	Ex40	15.4	±0.4	44.3	±1.3	6.9	±0.2
	Ex80	15.5	±0.5	44.9	±1.4	7.0	±0.2
	Ex120	15.6	±0.5	45.0	±1.3	7.1	±0.2

Values = Mean ± SE; Abbreviations, Eu=euhydration, GD=Glycerol hyperhydration with no rehydration, GR=Glycerol hyperhydration with rehydration, WD=Water hyperhydration with no rehydration, WR=Water hyperhydration with rehydration, Ex0=pre-exercise, Ex40=40 min of exercise, Ex80= 80 min of exercise,
Ex120 = 120 min of exercise

FIGURE 3

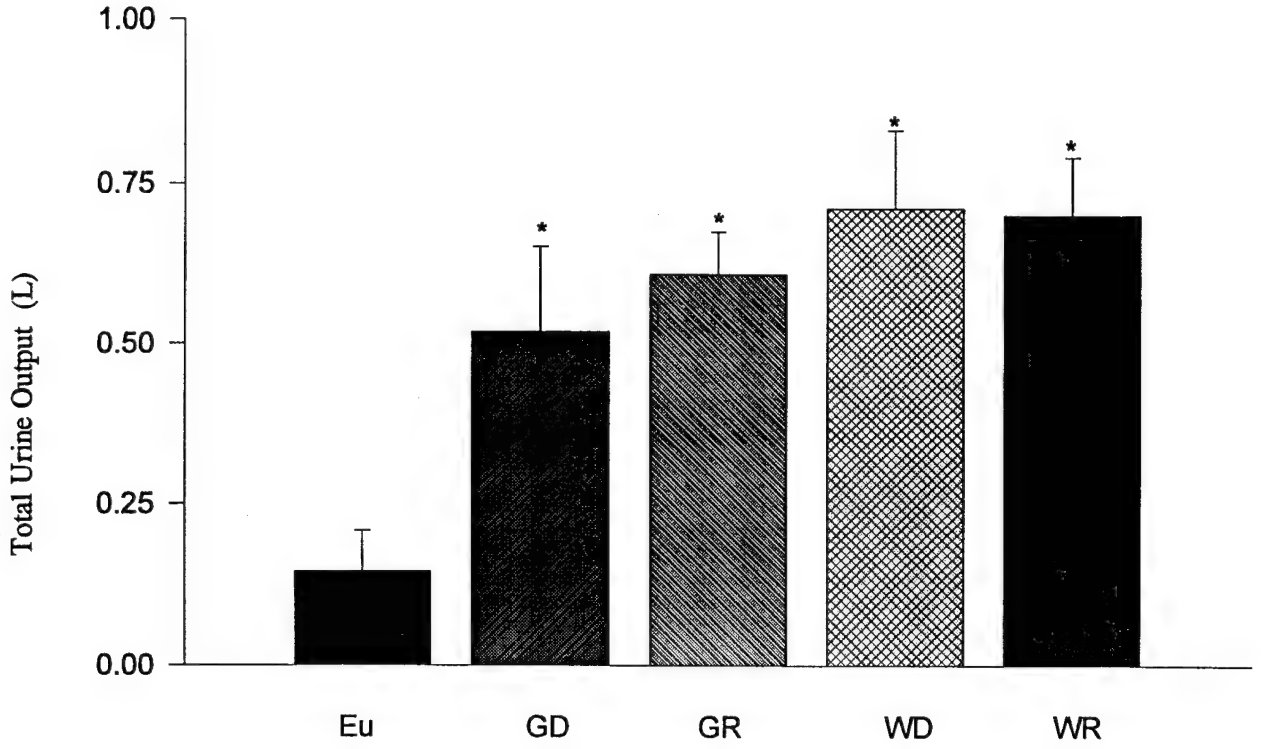

FIGURE 3: Total urine volumes during compensable exercise-heat stress trials. Values are means ± SE; Eu = euhydration, GD = glycerol hyperhydration with no rehydration, GR = glycerol hyperhydration with rehydration, WD = water hyperhydration with no rehydration, WR = water hyperhydration with rehydration. *Significantly different ($P<0.05$) from Eu trial.

FIGURE 4

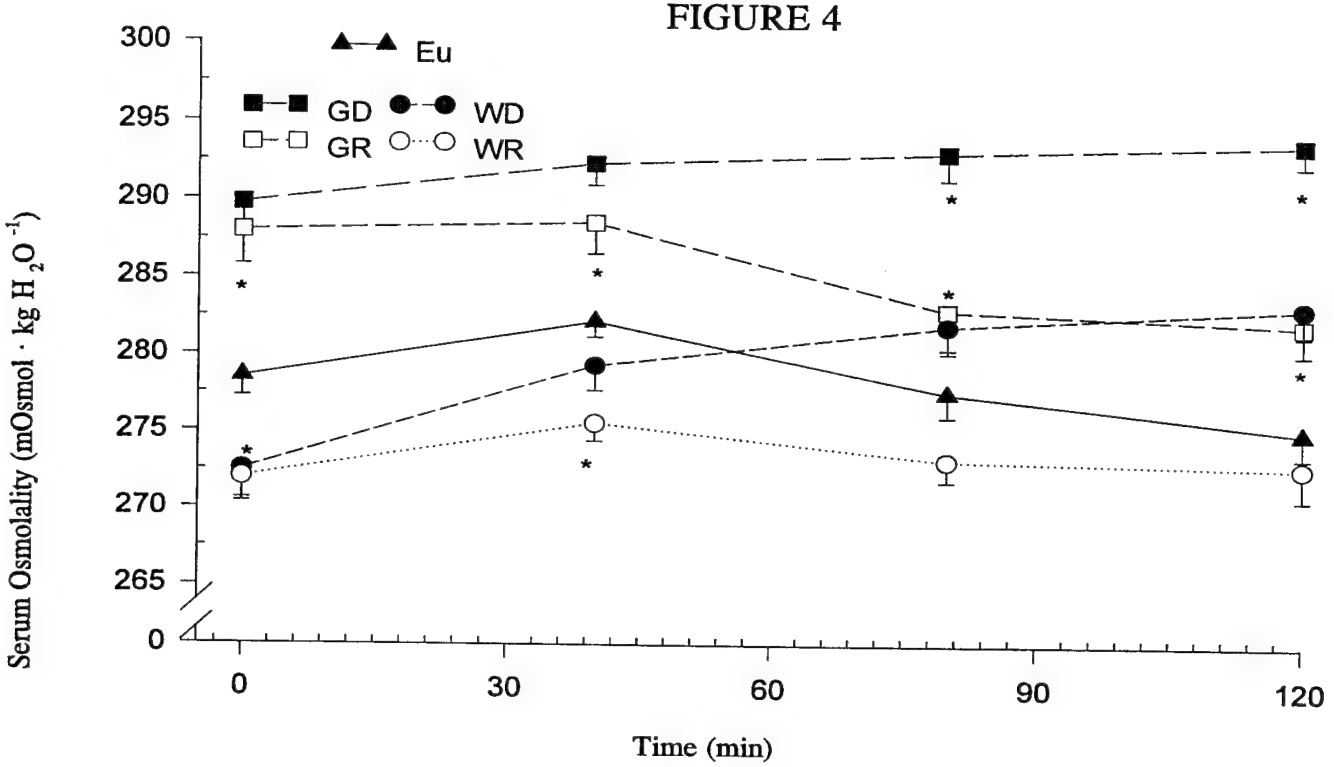

FIGURE 4: Serum osmolalities during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration.

Table 7 presents serum glycerol values during each trial. Serum glycerol levels were greater ($P<0.05$) in GD and GR than in Eu, WD and WR trials. During exercise serum glycerol did not change ($P>0.05$) in Eu, WD, and WR trials; however, serum glycerol decreased ($P<0.05$) during exercise in GD and GR by 39 and 31 $mg \cdot dl^{-1}$, respectively.

Table 8 presents serum values for sodium, potassium and lactate during each trial. Pre-exercise and during exercise, serum lactate and serum potassium levels were similar ($P>0.05$) during each trial. Serum sodium was similar ($P>0.05$) in all trials during pre-exercise, but during exercise values increased ($P<0.05$) in GD and WD.

Metabolic Rate and Heart Rate

Figure 5 presents the metabolic rates during exercise-heat stress for each trial. Metabolic rate responses increased ($P<0.05$) over time during exercise, but were not different ($P>0.05$) at any time between trials. The average metabolic rates were 337±48, 342±43, 338±48, 341±45, and 346±51 $W \cdot m^{-2}$, which corresponded to relative intensity oxygen uptake levels of 44%, 45%, 44%, 45% and 45% of $\dot{V}O_{2\ max}$, for Eu, GD, GR, WD, and WR trials, respectively.

Figure 6 presents the heart rate responses during each trial. Heart rate was not different ($P>0.05$) at rest between trials, and increased ($P<0.05$) over time during exercise. Final exercise heart rate values were greater ($P<0.05$) in GD and WD trials (158±9 and 161±15 $beats \cdot min^{-1}$, respectively) than in GR and WR trials (149±13 and 148±17 $beats \cdot min^{-1}$, respectively). Final exercise heart rates were similar ($P>0.05$) in GR, WR and Eu trials (14 ±13, 148±17 and 150±14 $beats \cdot min^{-1}$, respectively).

Table 7. Mean ± SE Serum Osmolality and Serum Glycerol

Trial	Time	Osmolality ($mOsmol \cdot kg^{-1}$)		Glycerol ($mg \cdot dl^{-1}$)	
Eu	Ex0	279	±1	0.3	±0.2
	Ex40	282	±1	1.6	±0.3
	Ex80	278	±2	2.5	±0.3
	Ex120	275	±2	3.8	±0.4
GD	Ex0	290	±2	121.6	±5.4
	Ex40	292	±1	103.5	±4.7
	Ex80	293	±2	93.0	±6.1
	Ex120	294	±1	84.4	±7.7
GR	Ex0	288	±2	112.5	±6.0
	Ex40	289	±2	105.8	±3.9
	Ex80	283	±3	92.6	±5.7
	Ex120	282	±2	82.7	±5.9
WD	Ex0	273	±2	0.0	±0.0
	Ex40	279	±2	1.3	±0.2
	Ex80	282	±2	2.0	±0.3
	Ex120	283	±2	3.4	±0.3
WR	Ex0	272	±2	0.1	±0.1
	Ex40	276	±1	1.3	±0.2
	Ex80	273	±1	2.1	±0.3
	Ex120	273	±2	3.3	±0.3

Values = Mean ± SE; Abbreviations, Eu=euhydration, GD=Glycerol hyperhydration with no rehydration, GR=Glycerol hyperhydration with rehydration, WD=Water hyperhydration with no rehydration, WR=Water hyperhydration with rehydration, Ex0=pre-exercise, Ex40 = 40 min of exercise, Ex80= 80 min of exercise,
Ex120 = 120 min of exercise

Table 8. Mean ±SE Serum Values Taken Before and During HSTs

Trial	Time	Sodium ($mEq \cdot L^{-1}$)		Potassium ($mEq \cdot L^{-1}$)		Lactate ($mmol \cdot L^{-1}$)	
Eu	Ex0	136.0	±0.8	4.0	±0.1	1.5	±0.1
	Ex40	138.0	±0.7	4.5	±0.1	1.7	±0.2
	Ex80	136.0	±0.7	4.5	±0.1	1.8	±0.2
	Ex120	135.0	±1.0	4.6	±0.1	1.8	±0.1
GD	Ex0	132.8	±0.9	4.0	±0.1	1.8	±0.1
	Ex40	135.3	±0.9	4.5	±0.1	1.7	±0.2
	Ex80	137.3	±1.0	4.5	±0.1	1.6	±0.1
	Ex120	138.5	±1.1	4.6	±0.1	1.7	±0.1
GR	Ex0	132.7	±1.2	4.0	±0.1	1.8	±0.1
	Ex40	134.0	±1.0	4.5	±0.1	1.4	±0.1
	Ex80	132.5	±1.3	4.5	±0.1	1.6	±0.1
	Ex120	131.7	±1.4	4.6	±0.2	1.7	±0.1
WD	Ex0	134.4	±1.3	4.0	±0.1	1.6	±0.1
	Ex40	136.8	±1.1	4.5	±0.1	1.7	±0.2
	Ex80	137.6	±1.4	4.5	±0.1	1.8	±0.2
	Ex120	137.8	±1.6	4.7	±0.1	2.0	±0.2
WR	Ex0	134.1	±1.0	4.0	±0.1	1.7	±0.1
	Ex40	134.8	±1.1	4.4	±0.1	1.7	±0.2
	Ex80	134.3	±1.3	4.4	±0.1	1.9	±0.2
	Ex120	133.3	±1.4	4.4	±0.2	2.0	±0.2

Values = Mean ±SE; Abbreviations, Eu=euhydration, GD=Glycerol hyperhydration with no rehydration, GR=Glycerol hyperhydration with rehydration, WD=Water hyperhydration with no rehydration, WR=Water hyperhydration with rehydration, Ex0=pre-exercise, Ex40 at ~40 min of exercise, Ex80= 80 min of exercise, Ex120 = 120 min of exercise

FIGURE 5

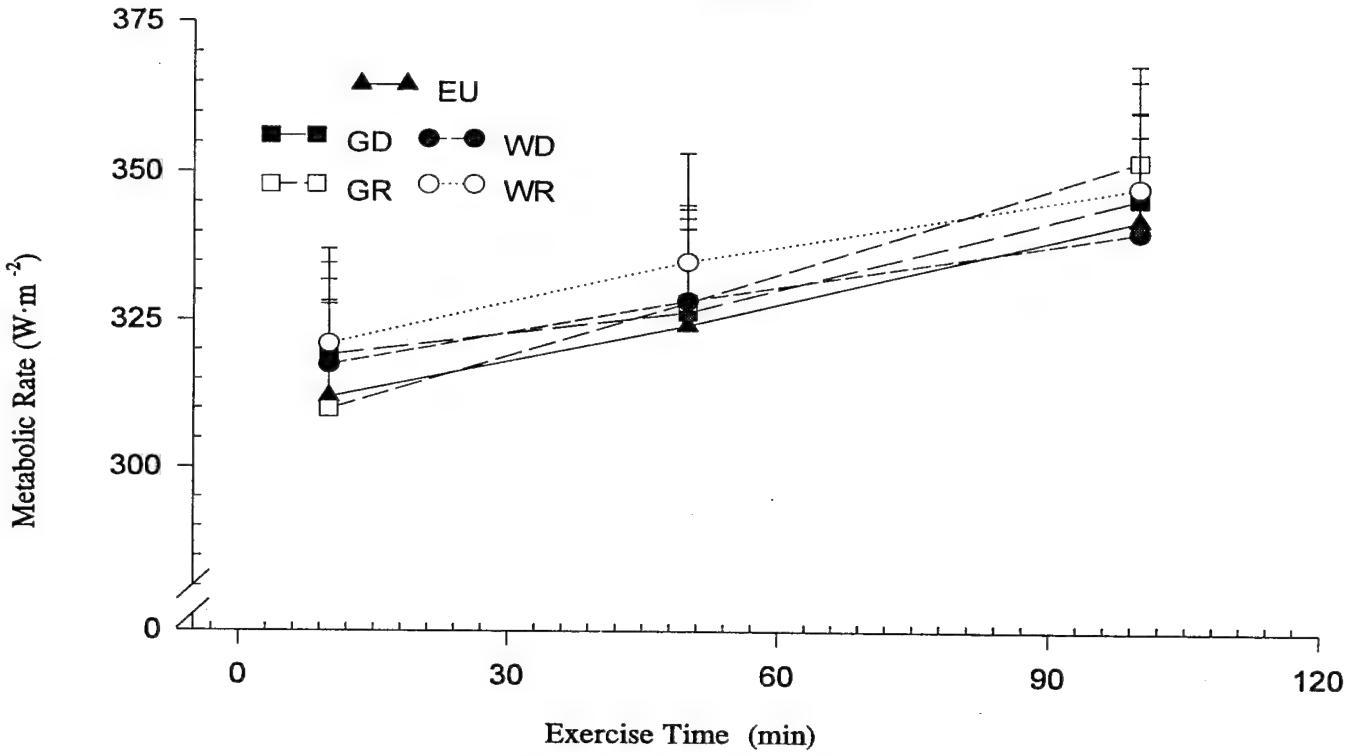

FIGURE 5: Metabolic rates during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration.

FIGURE 6

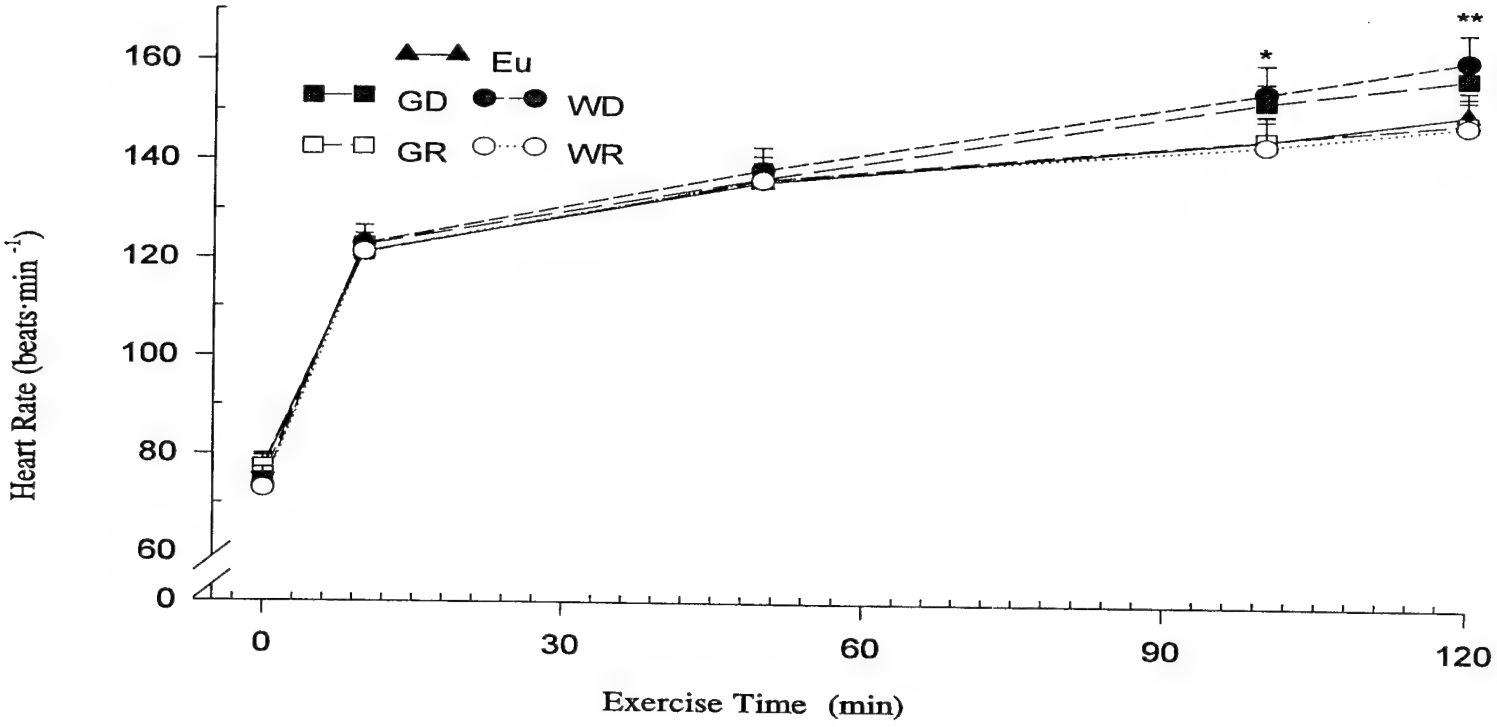

FIGURE 6: Heart rate responses during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration. *Heart rates are greater ($P<0.05$) in GD and WD than WR. **Heart rates are greater ($P<0.05$) in GD and WD than GR, WR, and Eu trials.

Body Temperature

Figures 7 and **8** present the rectal temperature and esophageal temperature responses during each trial, respectively. Rectal temperature (T_{re}) values were not different ($P>0.05$) between trials either pre-exercise or at any time during exercise, and values increased ($P<0.05$) over time during exercise. Final exercise T_{re} values were 38.6±0.4, 38.8±0.2, 38.5±0.3, 38.7±0.4 and 38.6±0.4 °C, for Eu, GD, GR, WD, and WR trials, respectively. The pre-exercise esophageal temperature (T_{es}) values were not different ($P>0.05$) between trials, and temperatures increased ($P<0.05$) over time during exercise. Final exercise T_{es} values were greater ($P<0.05$) during GD and WD (38.3±0.2 and 38.2±0.2 °C, respectively) than Eu (38.0±0.2°C) trials; however, similar ($P>0.05$) final values (38.1±0.2, 38.0±0.1 and 38.0±0.2 °C) were found during Eu, GR and WR trials, respectively.

Figure 9 presents mean skin temperatures ($\bar{T}_{sk}$) responses during each trial. The $\bar{T}_{sk}$ values were similar ($P>0.05$) between both pre-exercise and during exercise. Final exercise $\bar{T}_{sk}$ values for Eu, GD, GR, WD and WR trials were 35.4±0.9, 35.6±1.0, 35.3±1.3, 35.4±1.0, and 35.5±0.9 °C, respectively. **Figure 10** presents the core to skin temperature gradient (T_{re}-$\bar{T}_{sk}$) during each trial. The T_{re}-$\bar{T}_{sk}$ gradients increased ($P<0.05$) during exercise, but values were similar ($P>0.05$) between trials.

Figure 11 presents the mean body temperature ($\bar{T}_{b}$) responses during exercise for each trial. Mean body temperature values were not different ($P>0.05$) between trials either at pre-exercise or at any time during exercise, and temperatures were increased ($P<0.05$) during exercise. Final exercise $\bar{T}_{b}$ values were 38.2±0.1, 38.5±0.1, 38.1±0.1, 38.4±0.1 and 38.3±0.1 °C, for trials Eu, GD, GR, WD and WR, respectively.

Sweating Response

Figure 12 presents whole body sweating rate responses during exercise for each trial. Whole body sweating rates were not different ($P>0.05$) between trials. The whole body sweating rates were 529±50, 497±48, 520±67, 490±49 and 524±52 $g \cdot m^{-2} \cdot h^{-1}$, for trials Eu, GD, GR, WD and WR, respectively.

FIGURE 7

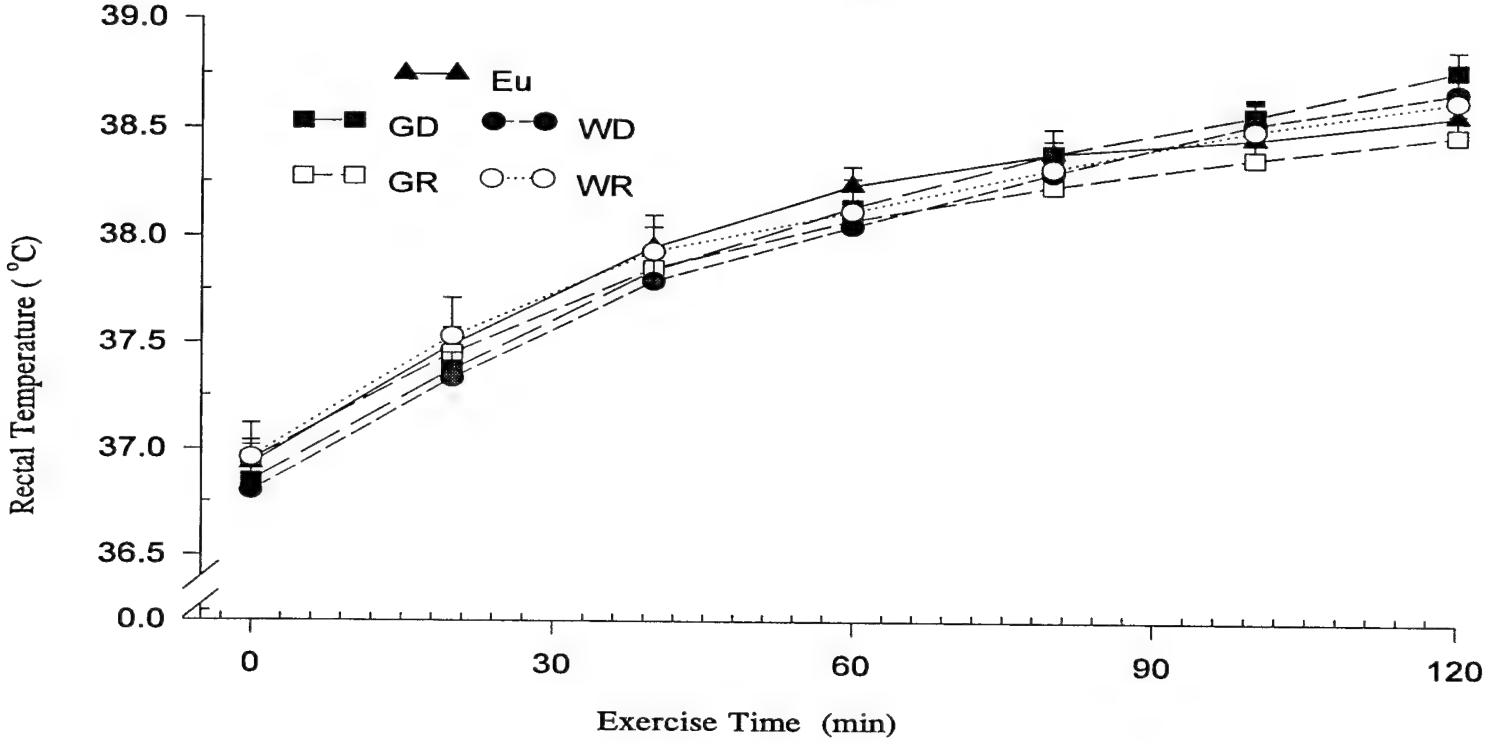

FIGURE 7: Rectal temperatures during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration.

FIGURE 8

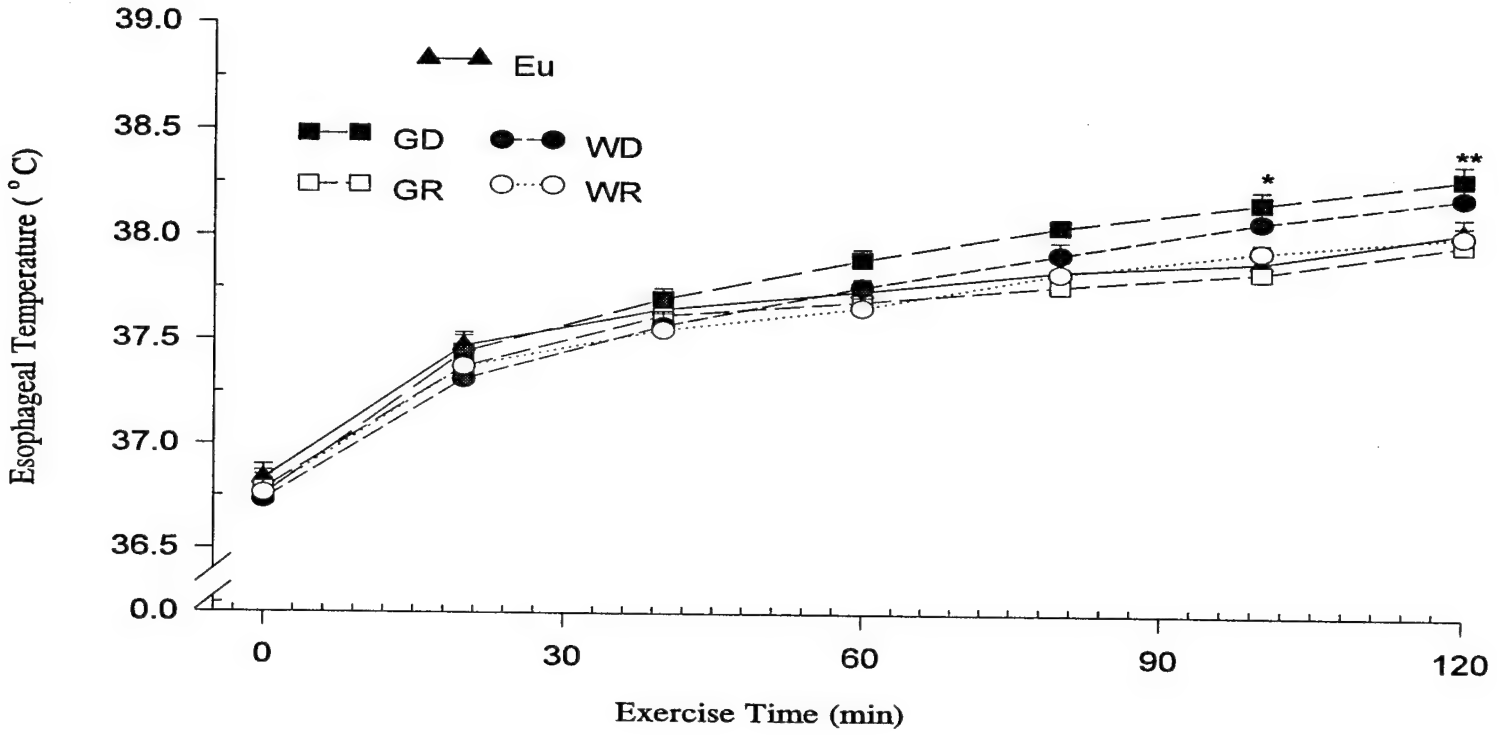

FIGURE 8: Esophageal temperatures during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration. *GD temperatures are greater ($P<0.05$) than GR and Eu. **GD and WD values are greater ($P<0.05$) than GR, WR and Eu.

FIGURE 9

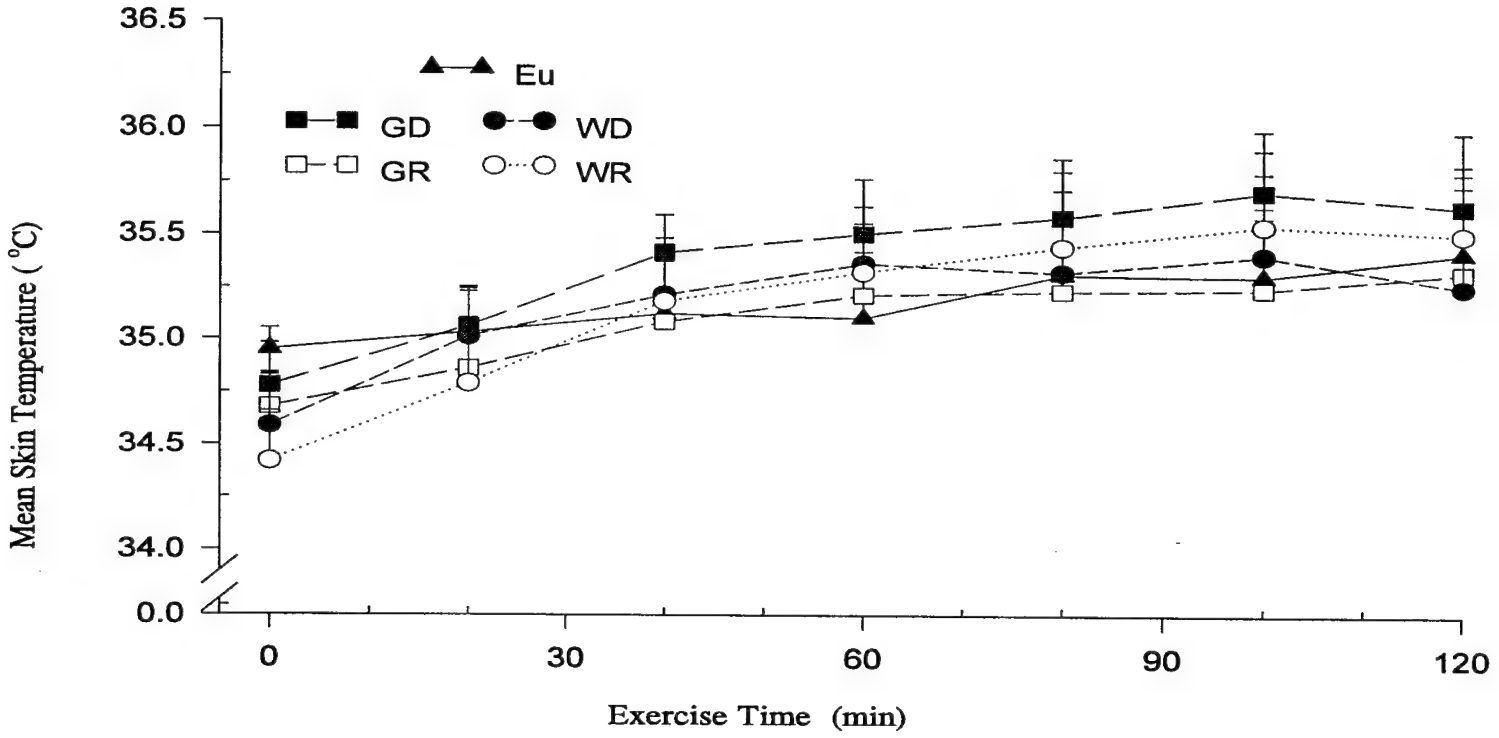

FIGURE 9: Mean skin temperatures during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration.

FIGURE 10

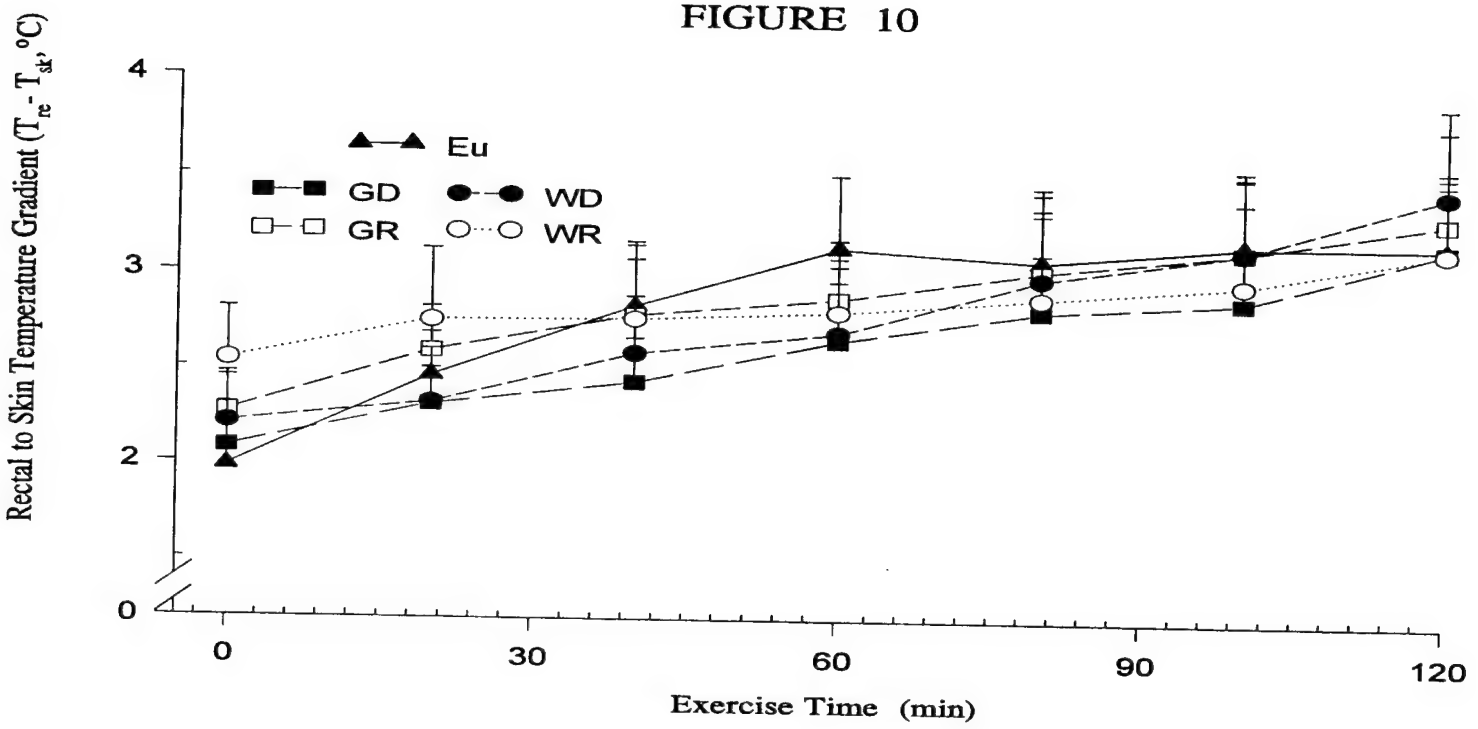

FIGURE 10: Rectal to skin temperature gradients during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration.

FIGURE 11

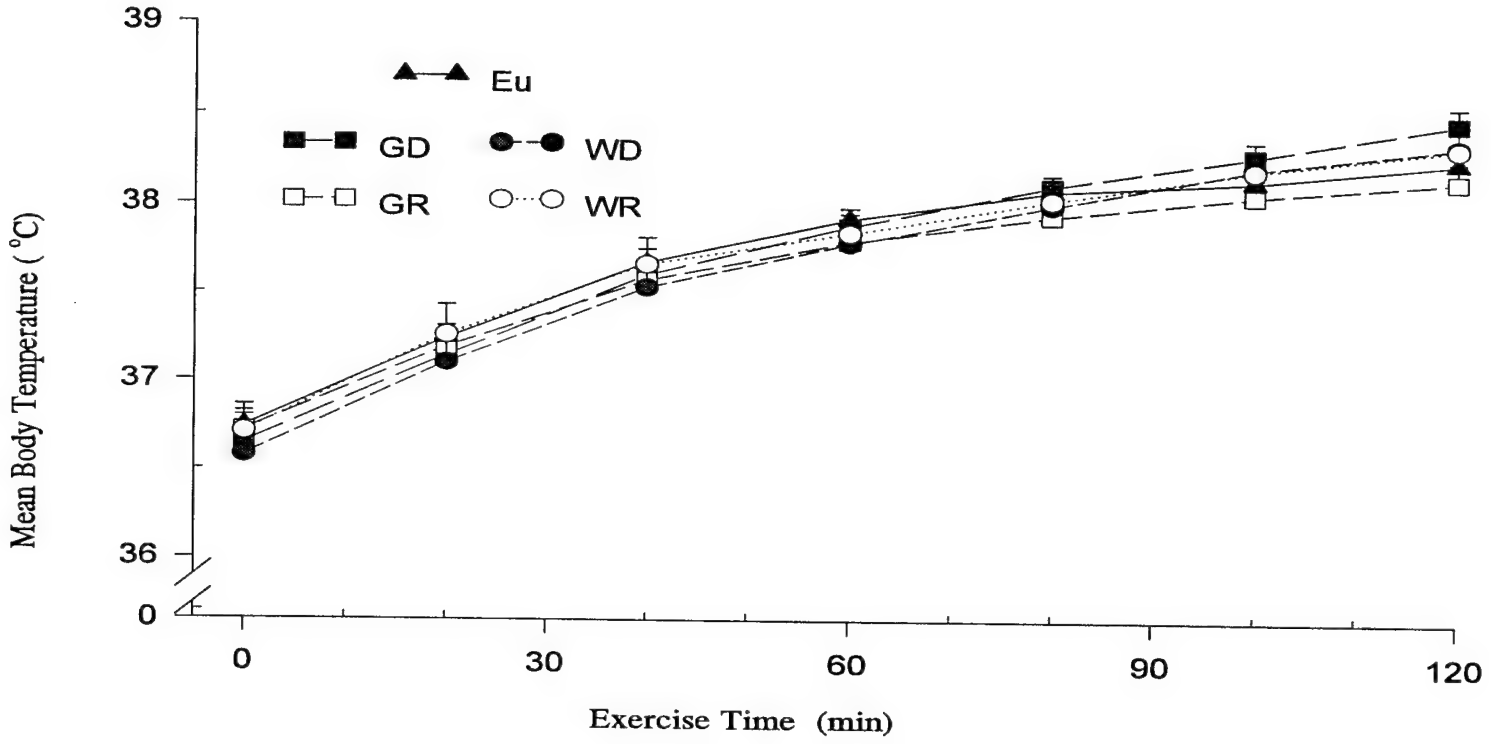

FIGURE 11: Mean body temperatures during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration.

FIGURE 12

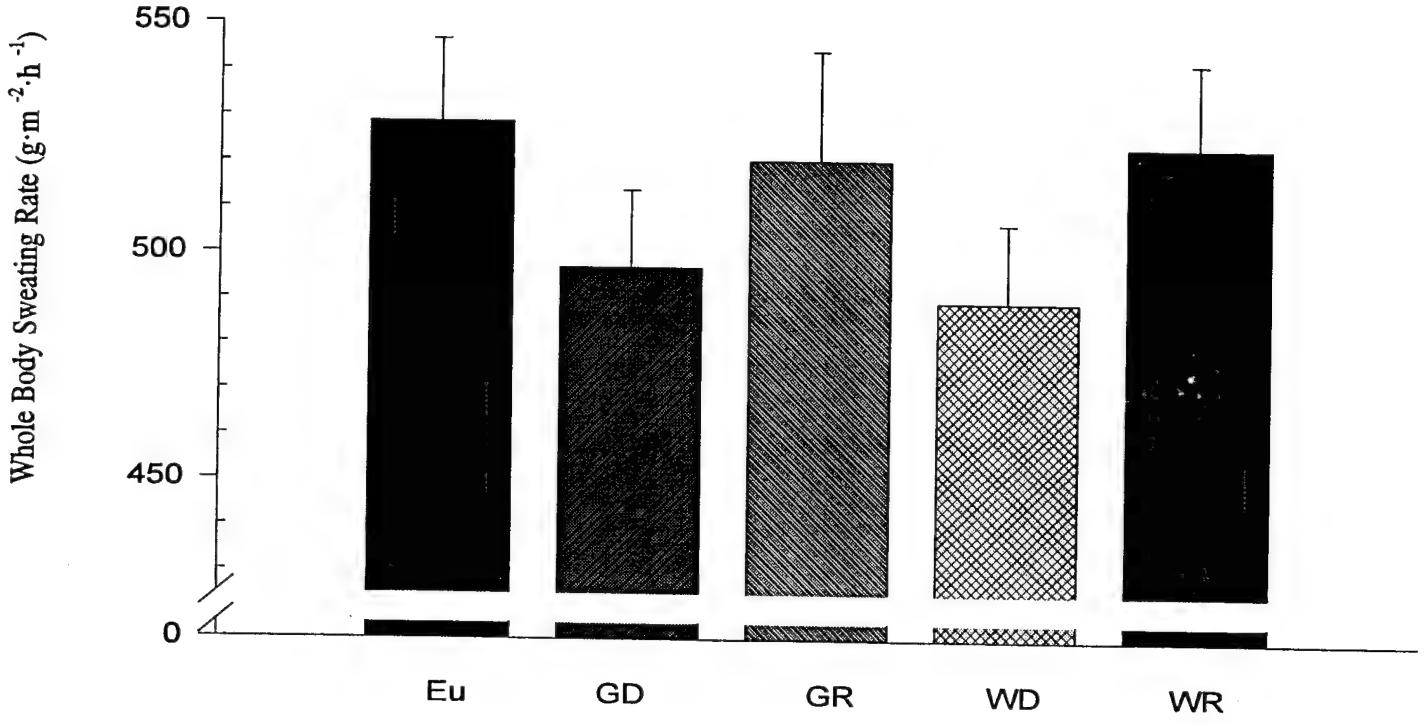

FIGURE 12: Whole body sweating rates during compensable exercise-heat stress trials. Values are means ± SE; Eu = euhydration, GD = glycerol hyperhydration with no rehydration, GR = glycerol hyperhydration with rehydration, WD = water hyperhydration with no rehydration, WR = water hyperhydration with rehydration.

Figure 13 presents local sweating rates every 20 min during exercise for each trial. Local sweating rates were not different ($P>0.05$) between trials. Final local sweating rates were 1.09±0.20, 1.08±0.23, 1.00±0.28, 0.99±0.29, and 1.05±0.28 $mg \cdot cm^{-2} \cdot min^{-1}$ for trials Eu, GD, GR, WD and WR, respectively. **Figure 14** presents local sweating threshold temperatures for each trial. Sweating threshold temperature values were not different $P>0.05$ between trials. Sweating threshold temperatures were 36.7±0.2, 36.5±0.4, 36.5±0.1, 36.5±0.3 and 36.5±0.4 °C, for trials Eu, GD, GR, WD and WR, respectively. **Figure 15** presents local sweating sensitivity values for each trial. Local sweating sensitivities were not different ($P>0.05$) between trials. Sweating sensitivity values were 1.01±0.30, 1.03±0.60, 1.03±0.41, 0.98±0.42 and 0.96±0.53 $mg \cdot cm^{-2} \cdot min^{-1} \cdot °C^{-1}$, for trials Eu, GD, GR, WD and WR, respectively.

PHASE II, UNCOMPENSABLE EXERCISE-HEAT STRESS

The eight subjects that completed five trials in Phase I, also completed the three trials in Phase II.

Preliminary Hydration Measurements

Prior to each trial, pre-drink body masses were similar ($P>0.05$) for each trial. For CON, GD and WD trials body masses were 76.0±15.1, 76.1±14.6 and 75.9±14.6 kg, respectively. The pre-drink plasma osmolalities were also similar ($P>0.05$) between trials. Plasma osmolalities for CON, GD and WD were 283±4, 285±4 and 284±3 mOsmol/kg H_2O, respectively.

Fluid Ingestion

For each subject the same volume of fluid was ingested before exercise in GD and WD trials, and no fluid was ingested in the CON trial. The volume of fluid consumed before each hyperhydration trial was 1.84±0.25 L. **Figure 16** presents change in total TBW during each trial. For GD and WD, TBW increased ($P<0.05$) by 1.50±0.41 and 1.42±0.32 L, respectively at 60 min after drinking with no difference ($P>0.05$) between trials. In all trials TBW decreased ($P<0.05$) during exercise.

FIGURE 13

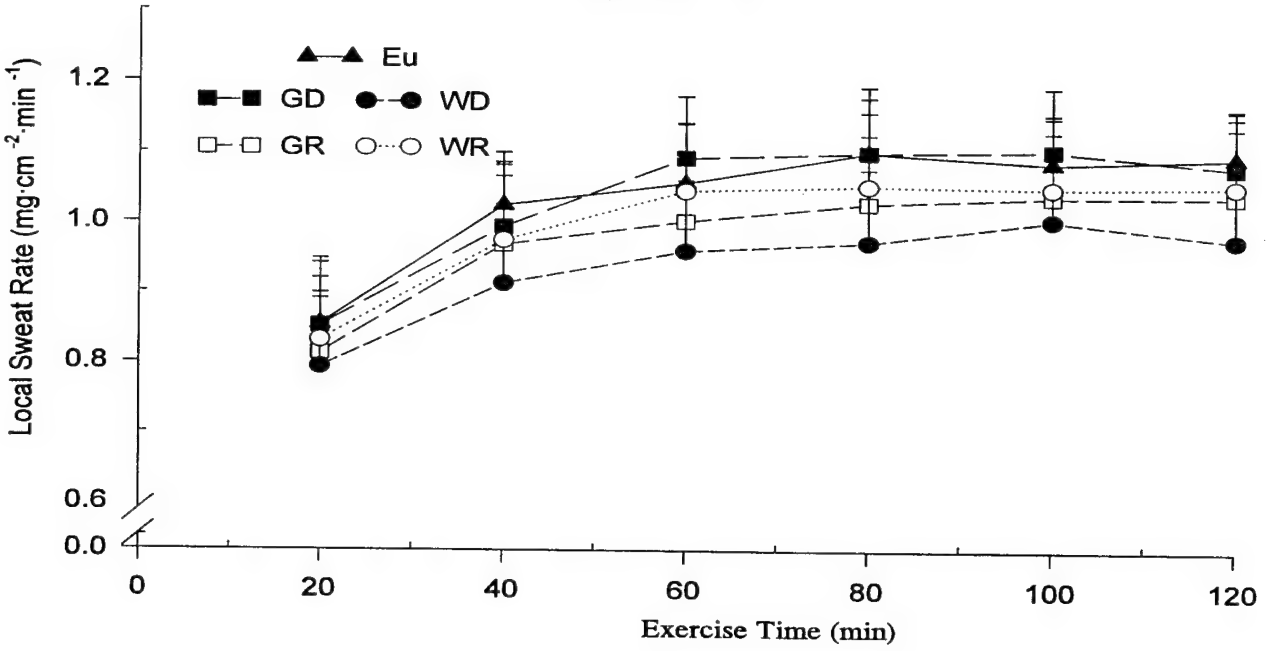

FIGURE 13: Local sweating rates during compensable exercise-heat stress trials. Values are means ± SE; Eu (filled triangles) = euhydration, GD (filled square) = glycerol hyperhydration with no rehydration, GR (open square) = glycerol hyperhydration with rehydration, WD (filled circles) = water hyperhydration with no rehydration, WR (open circle) = water hyperhydration with rehydration.

FIGURE 14

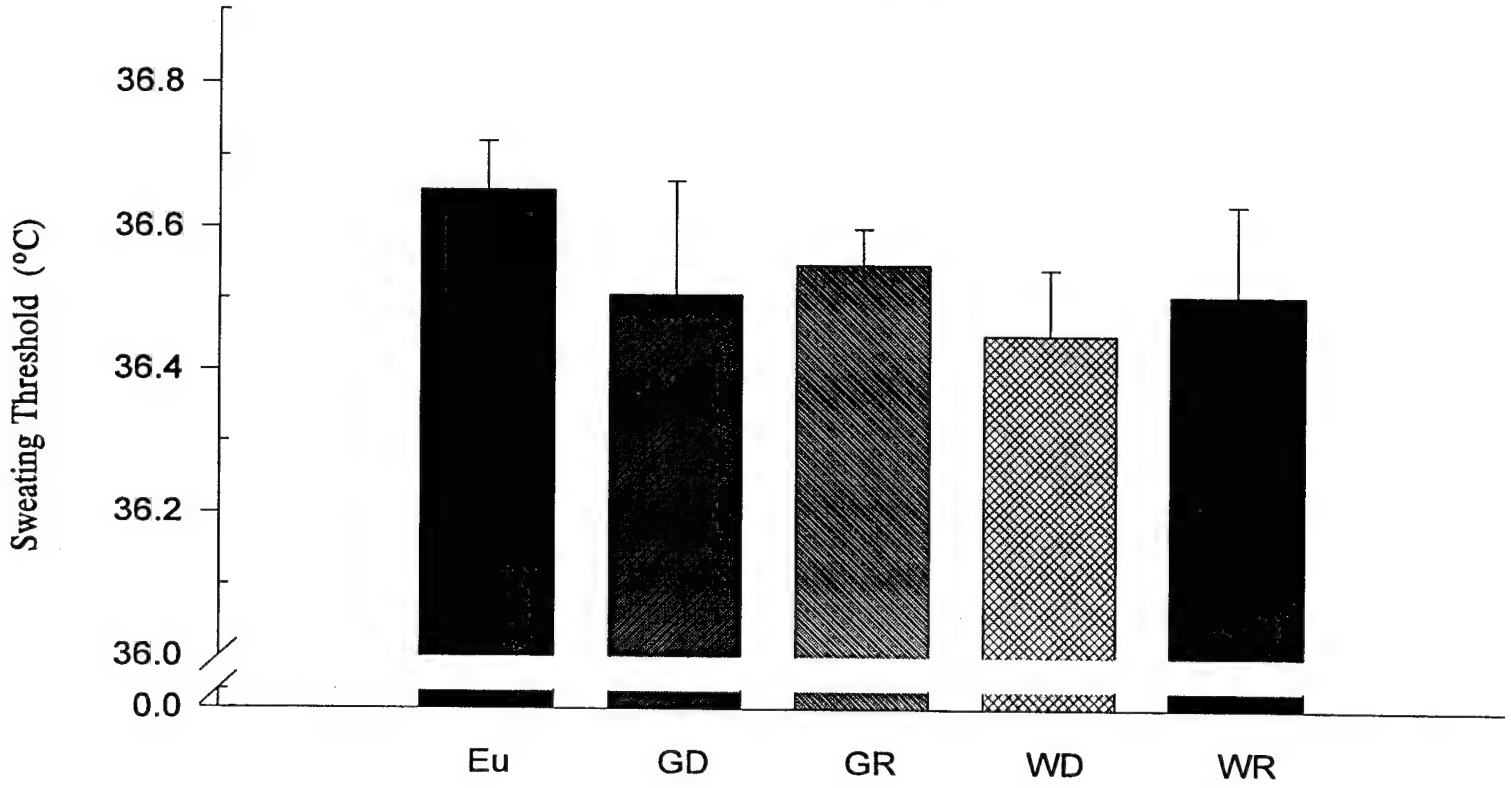

FIGURE 14: Sweating thresholds during compensable exercise-heat stress trials. Values are means ± SE; Eu = euhydration, GD = glycerol hyperhydration with no rehydration, GR = glycerol hyperhydration with rehydration, WD = water hyperhydration with no rehydration, WR = water hyperhydration with rehydration.

FIGURE 15

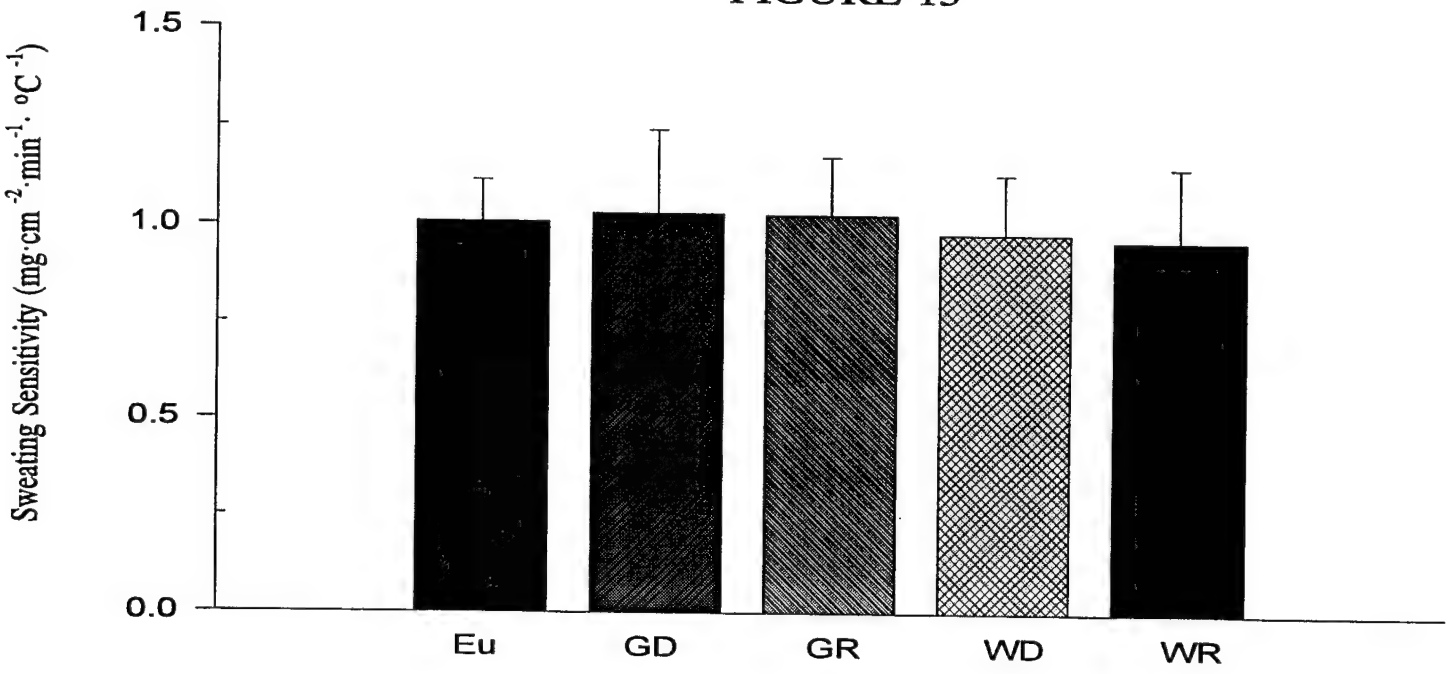

FIGURE 15: Local sweating sensitivities during compensable exercise-heat stress trials. Values are means ± SE; Eu = euhydration, GD = glycerol hyperhydration with no rehydration, GR = glycerol hyperhydration with rehydration, WD = water hyperhydration with no rehydration, WR = water hyperhydration with rehydration. GD and WD, respectively. The percent decreases in TBW from pre-drink to post exercise were 3.2±0.7%, 0.3±1.0% and 0.2±0.6% for trials CON, GD and WD, respectively. There were no differences ($P>0.05$) in TBW between GD and WD before or during exercise, but TBW was greater ($P<0.05$) in GD and WD trials than in the CON trial during exercise.

During exercise TBW values decreased 1.35±0.22, 1.63±0.34 and 1.64±0.23 L for trials CON, GD and WD, respectively. There was no difference ($P>0.05$) in TBW between GD and WD before or during exercise, but TBW was greater ($P<0.05$) in GD and WD than in CON trial during exercise.

Figure 17 presents total urine volume for each trial. Total urine volume was less ($P<0.05$) in the CON trial than in either GD or WD trials, and urine volumes were not different ($P>0.05$) between GD and WD trials. The total urine volumes were 0.11±0.12, 0.34±0.21 and 0.42±21 L, for CON, GD and WD trials, respectively.

Metabolic and Hemodynamic Responses

Figure 18 presents endurance time during uncompensable exercise-heat stress for each trial. The time to exhaustion was not different ($P>0.05$) between GD and WD trials; however, time to exhaustion was greater ($P<0.05$) in the GD trial than in the CON trial. Endurance times were 29.9±3.5, 33.8±3.0 and 31.3±3.1 min for CON, GD and WD trials, respectively. **Figure 19** presents the individual's variability for endurance time during uncompensable exercise-heat stress. Seven out of eight subjects had greater endurance times in the GD trials than the CON trials, and three out of eight subjects had greater endurance times in WD trials than CON trials. **Figure 20** presents the metabolic rate responses during exercise for each trial. Metabolic rate responses were not different ($P>0.05$) between trials or over time. The average metabolic rates were 414±23, 413±25 and 418±19 $W \cdot m^2$, for CON, GD and WD trials, respectively. These average metabolic rates correspond to a relative oxygen uptake intensity of 54%, 54% and 55% of $\dot{V}O_{2\ max}$, for CON, GD and WD trials, respectively.

Figure 21 presents heart rate responses during exercise for each trial. Heart rates were not different ($P>0.05$) at rest between trials and increased ($P<0.05$) over time during exercise. At 10 min of exercise heart rates were greater ($P<0.05$) in the CON trial than in GD and WD trials; however, as exercise progressed, this difference diminished. Heart rate responses during exercise were similar between GD and WD trials. Heart rates at exhaustion from heat strain were not different ($P>0.05$) between trials. These final heart rates were 187±12, 187±8 and 185±13 $beats \cdot min^{-1}$ for CON, GD and WD trials, respectively.

FIGURE 16

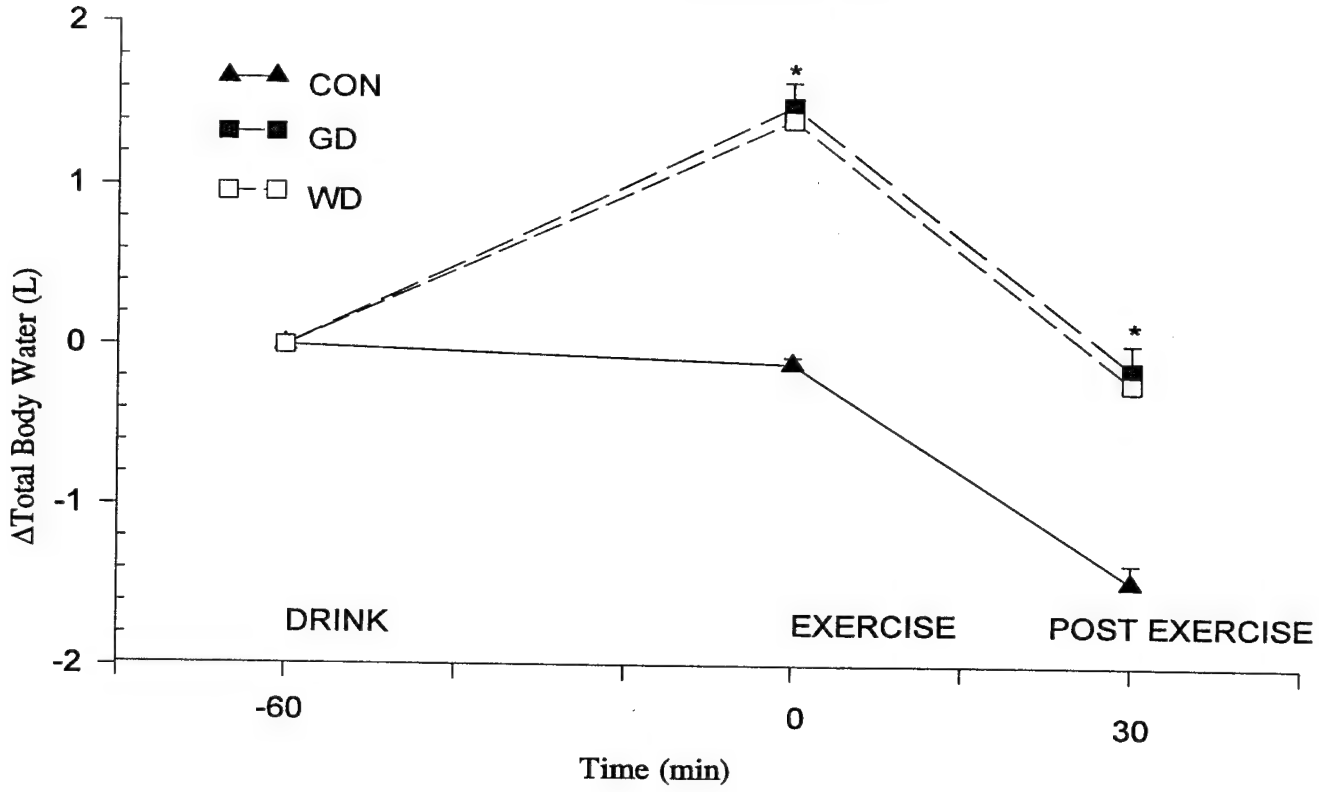

FIGURE 16: Changes in total body water over time during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration. *Significantly ($P<0.05$) different than CON.

FIGURE 17

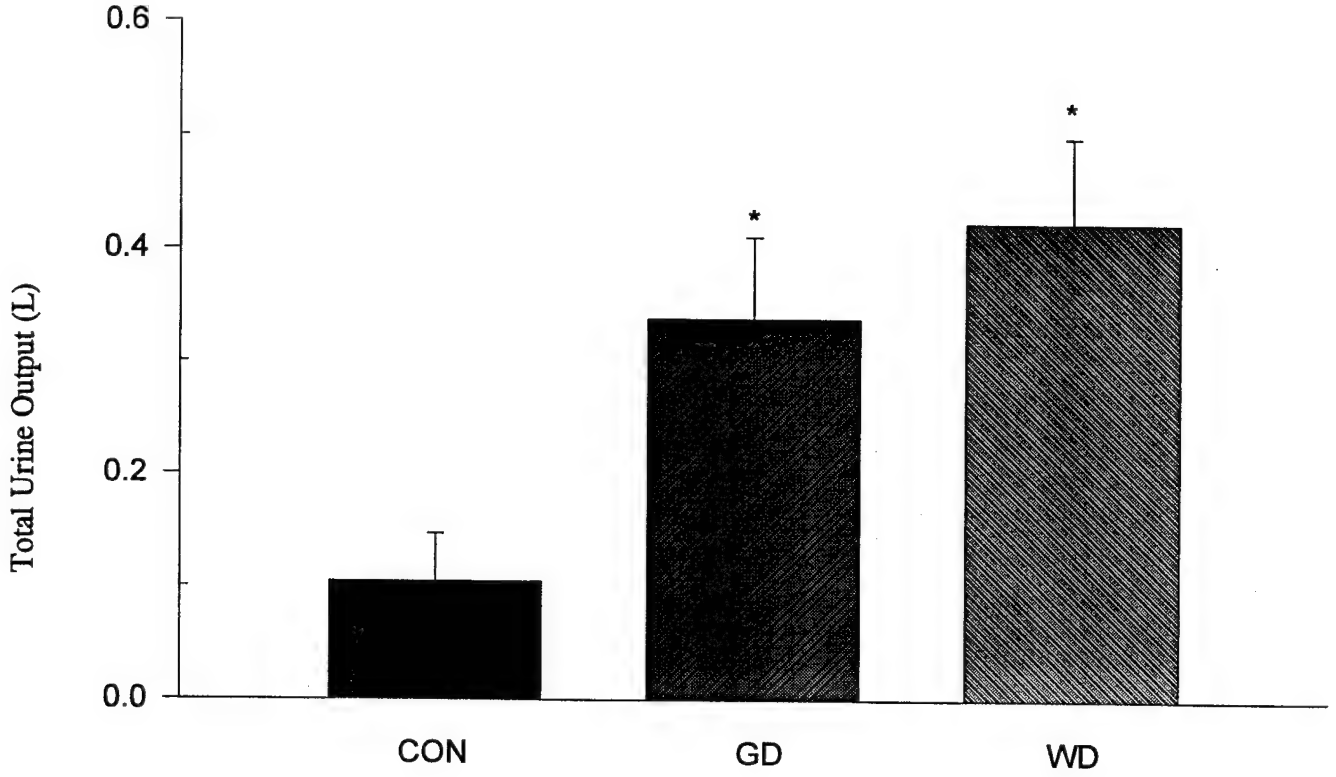

FIGURE 17: Total urine volumes during uncompensable exercise-heat stress trials. Values are means ± SE; CON = euhydration with no rehydration, GD = glycerol hyperhydration with no rehydration, WD = water hyperhydration with no rehydration. *Significantly ($P<0.05$) different than CON trial.

FIGURE 18

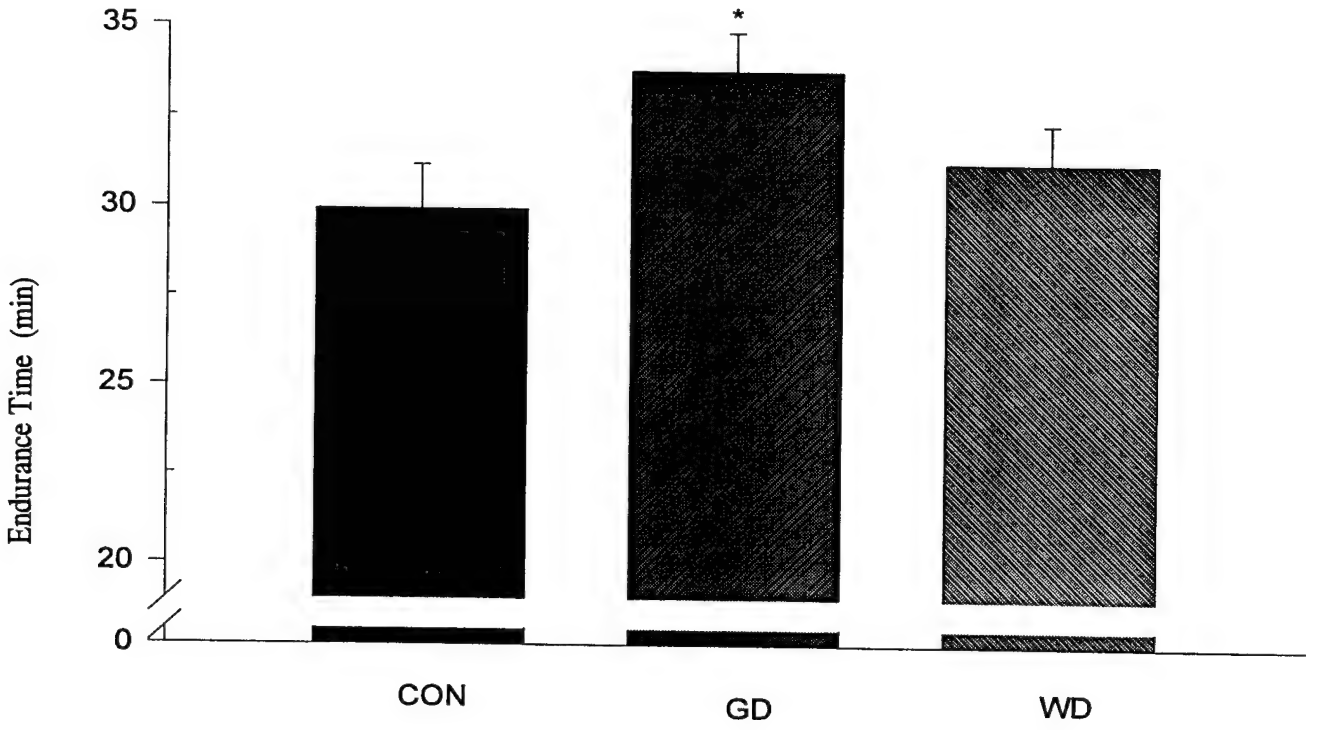

FIGURE 18: Endurance times for uncompensable exercise-heat stress trials. Values are means ± SE; CON = euhydration with no rehydration, GD = glycerol hyperhydration with no rehydration, WD = water hyperhydration with no rehydration. *Significantly ($P<0.05$) different than CON.

FIGURE 19

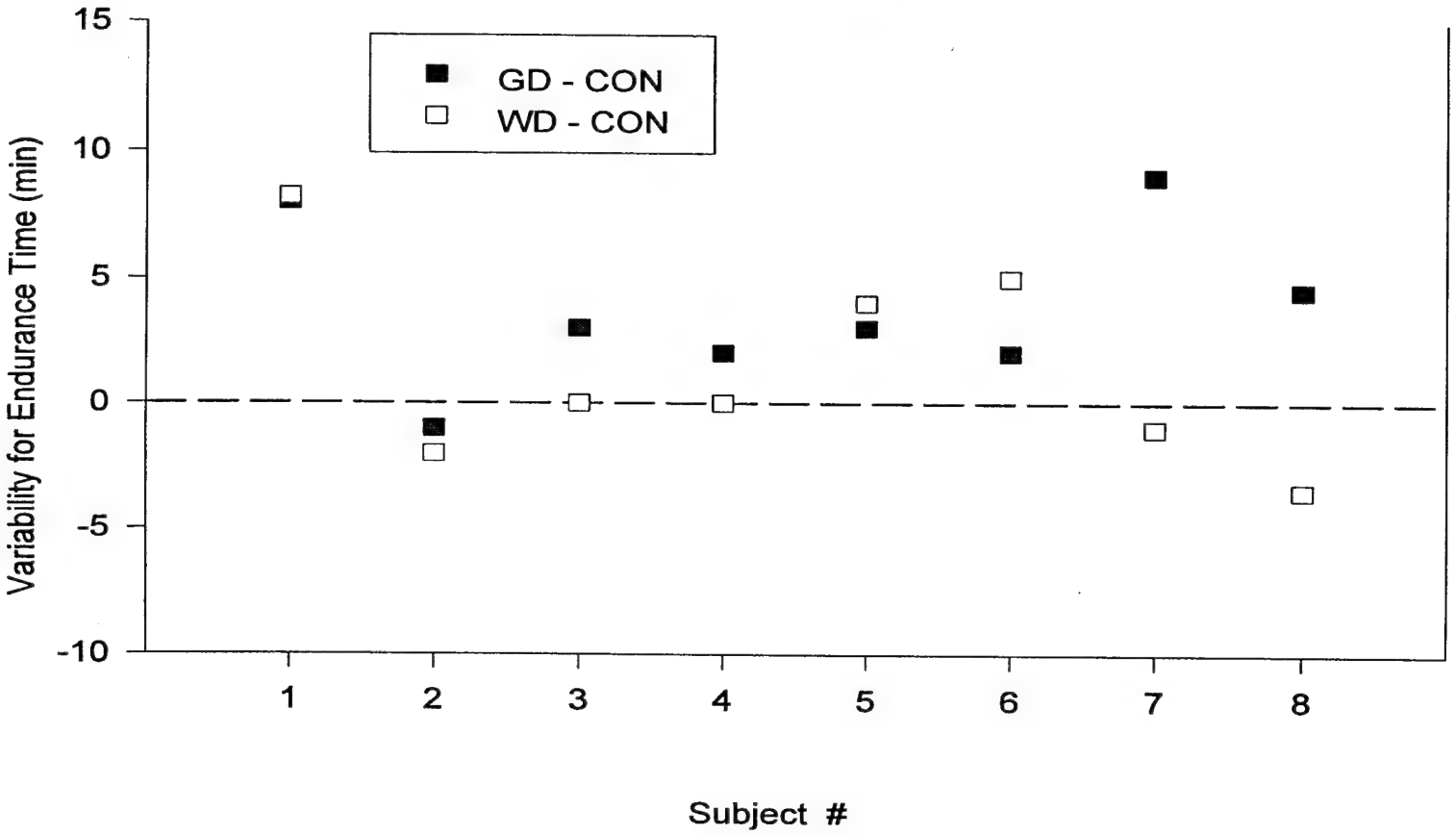

FIGURE 19: Individual's variability for endurance time during uncompensable exercise-heat stress trials. The values are GD and WD trials relative to CON trial, GD = glycerol hyperhydration with no rehydration, WD = water hyperhydration with no rehydration.

FIGURE 20

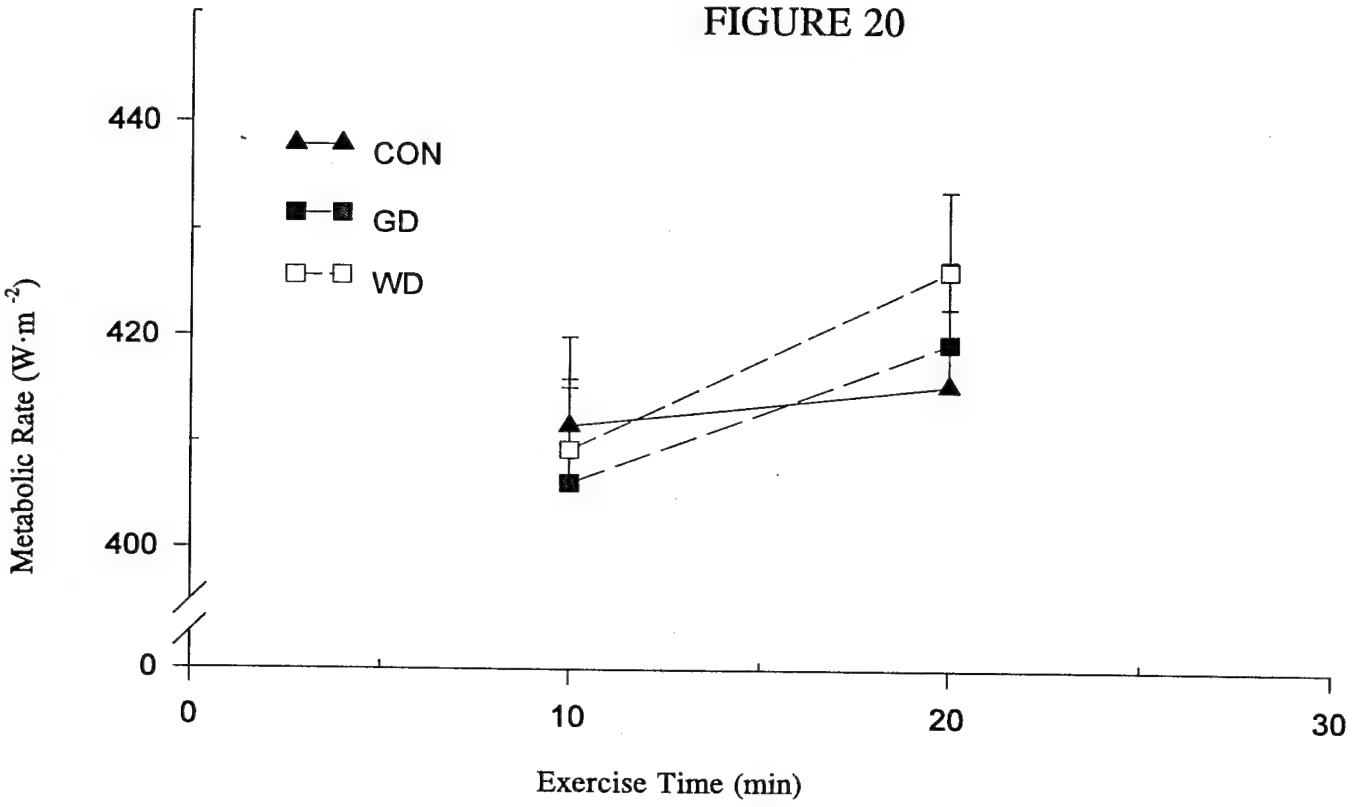

FIGURE 20: Metabolic rate responses during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration.

Figure 22 presents cardiac output during exercise for each trial. Cardiac output values were not different ($P>0.05$) between trials. The average cardiac output values during exercise were 17.1±3.1, 17.9±2.6 and 17.1±2.7 $L \cdot min^{-1}$ for CON, GD and WD trials, respectively. Cardiac output decreased ($P<0.05$) from 10 min to 20 min of exercise, and average values were 18.1±2.6 and 17.2±2.8 $L \cdot min^{-1}$, respectively.

Figure 23 presents the mean arterial pressure during exercise for each trial. Mean arterial pressure responses during rest or exercise were not different ($P>0.05$) between trials. Mean arterial pressure increased ($P<0.05$) over time during exercise. Final mean arterial pressure values were 98±14, 92±7 and 96±16 mmHg, for trials CON, GD and WD, respectively. **Figure 24** presents total peripheral resistance (TPR) during exercise for each trial. Total peripheral resistances were not different between trials. Final mean TPR values were 5.8±0.9, 5.5±0.9 and 5.8±0.7 PRU for trials CON, GD and WD, respectively.

Body Temperature and Sweating

Figure 25 presents the rectal temperature responses during exercise for each trial. Rectal temperature responses were not different ($P>0.05$) between trials either pre- or during exercise. Rectal temperatures increased ($P<0.05$) over time during exercise. Final T_{re} values were 38.7±0.4, 38.8±0.5 and 38.6±0.5 °C for CON, GD and WD trials, respectively.

Figure 26 presents mean skin temperature responses during exercise for each trial. The $\bar{T}_{sk}$ responses were similar ($P>0.05$) between trials both pre- and during exercise. The $\bar{T}_{sk}$ values were lower ($P<0.05$) at 5 min of exercise compared to pre-, and from 5 min of exercise, the $\bar{T}_{sk}$ values continued to increase ($P<0.05$) over time during exercise. Final exercise $\bar{T}_{sk}$ values for trials CON, GD and WD were 37.4±0.7, 37.6±0.7 and 37.1±1.1 °C, respectively. **Figure 27** presents the core to skin temperature gradient (T_{re}-$\bar{T}_{sk}$) during exercise for each trial. The T_{re}-$\bar{T}_{sk}$ gradient values were similar ($P>0.05$) between trials. The final exercise T_{re}-$\bar{T}_{sk}$ gradients were 1.3±1.0, 1.2±0.8 and 1.8±0.8 °C for CON, GD and WD trials, respectively. The 5 min exercise T_{re}-$\bar{T}_{sk}$ gradient values were greater ($P<0.05$) than pre-exercise values. After 5 min of exercise, the T_{re}-$\bar{T}_{sk}$ gradients decreased ($P<0.05$) over time during exercise.

FIGURE 21

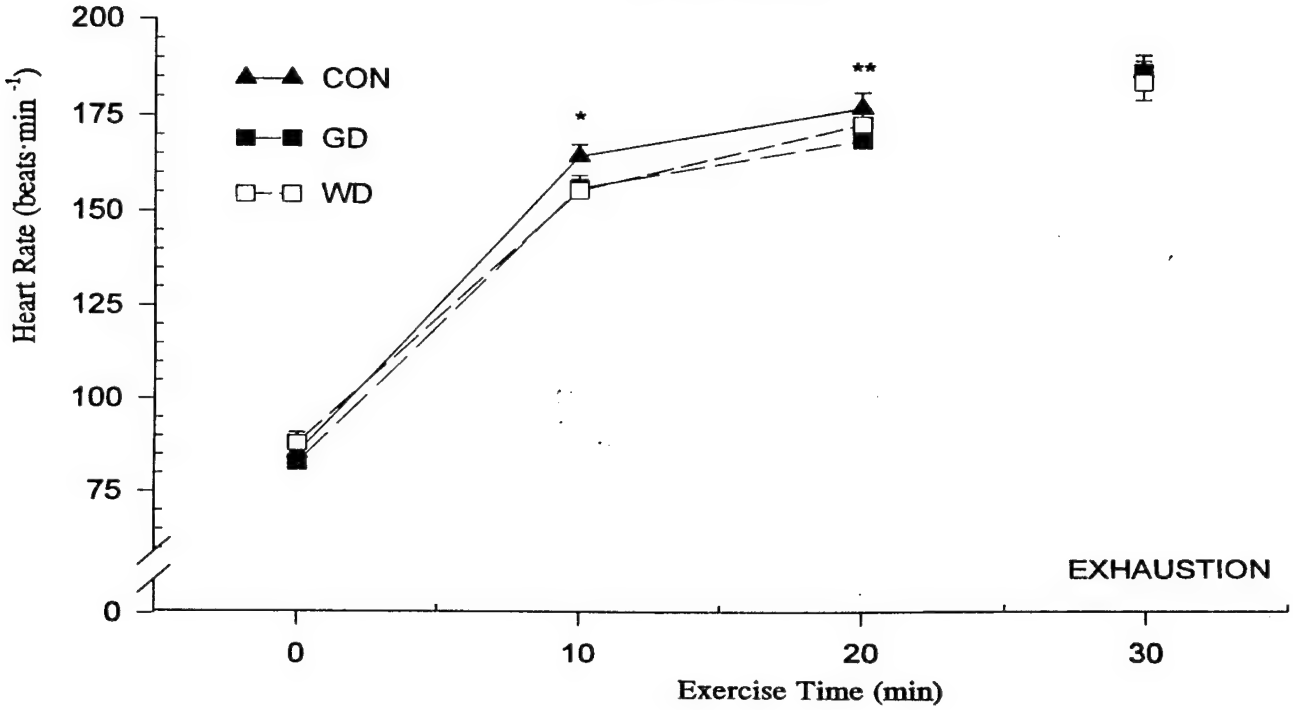

FIGURE 21: Heart rate responses during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration. * Trials GD and WD values are less ($P<0.05$) than CON. **Trial GD heart rate is lower ($P<0.05$) than CON trial.

FIGURE 22

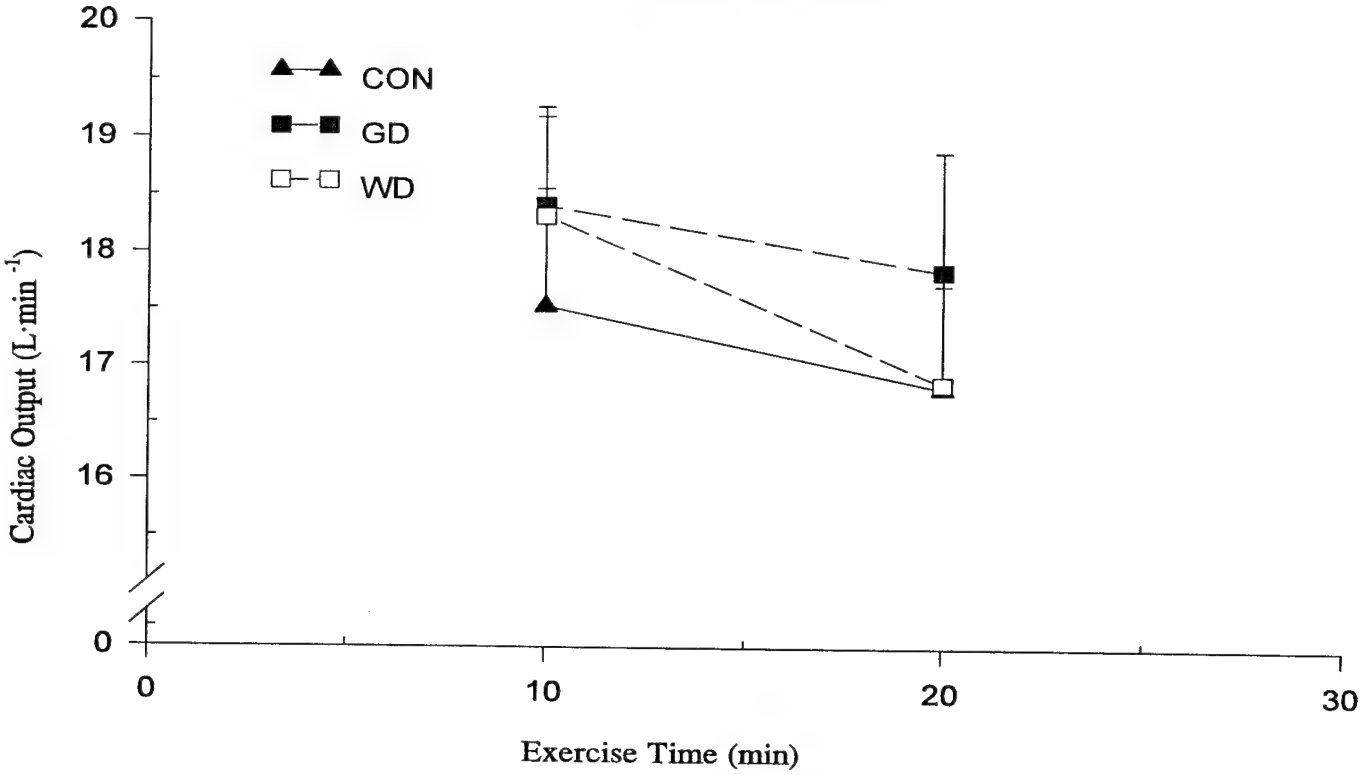

FIGURE 22: Cardiac output responses during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration. For all trials the 10-min value was greater ($P<0.05$) than the 20-min value.

FIGURE 23

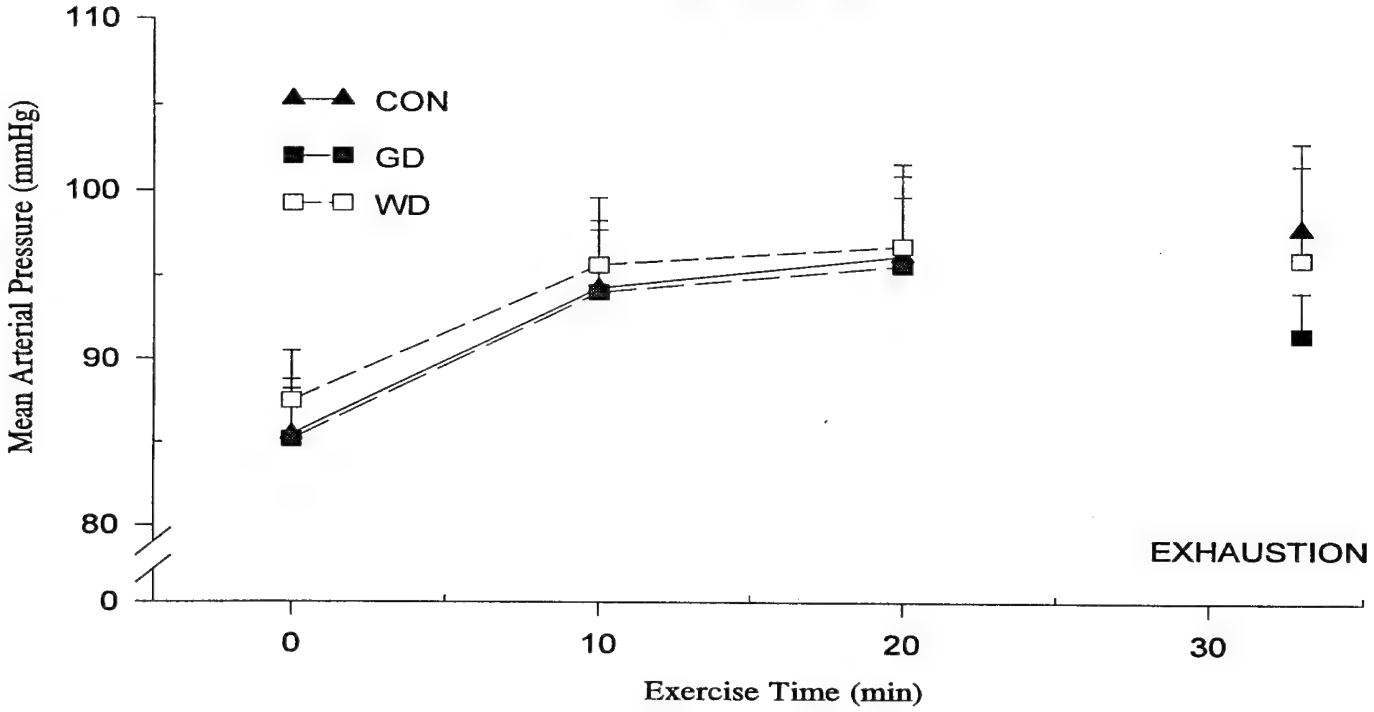

FIGURE 23: Mean arterial pressures during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration.

FIGURE 24

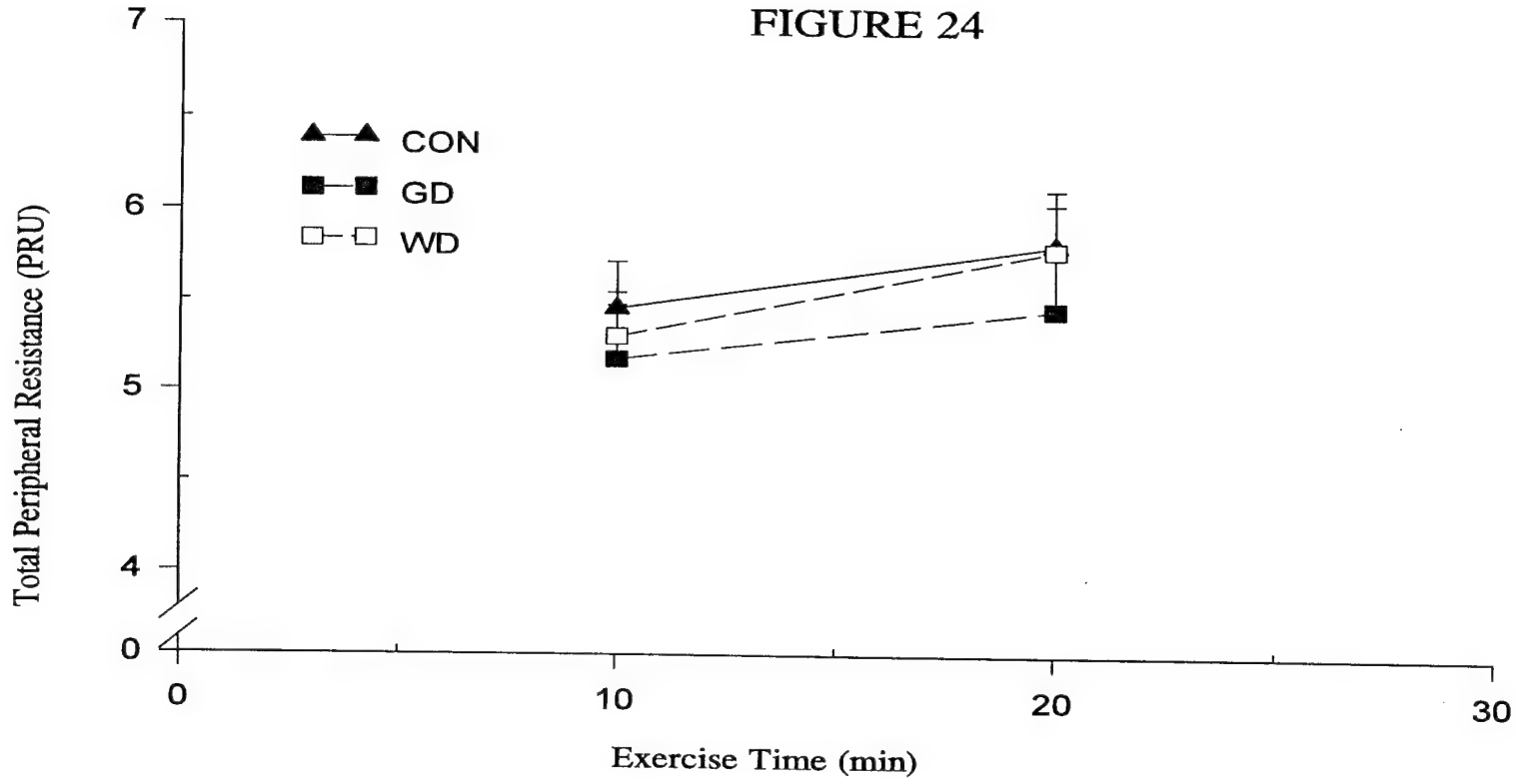

FIGURE 24: Total peripheral resistances during uncompensable exercise-heat stress trials. PRU = peripheral resistance units ($mmHg \cdot L^{-1} \cdot min^{-1}$) Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration.

FIGURE 25

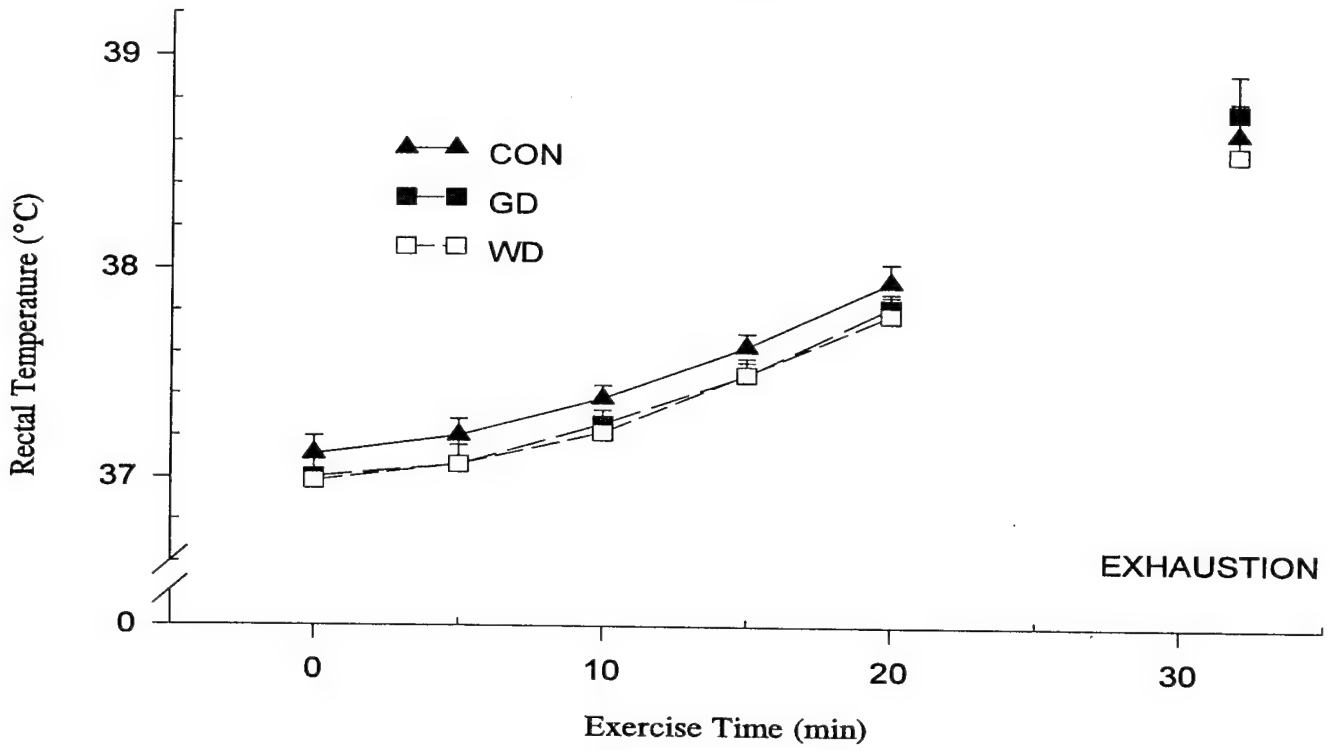

FIGURE 25: Rectal temperature values during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration.

FIGURE 26

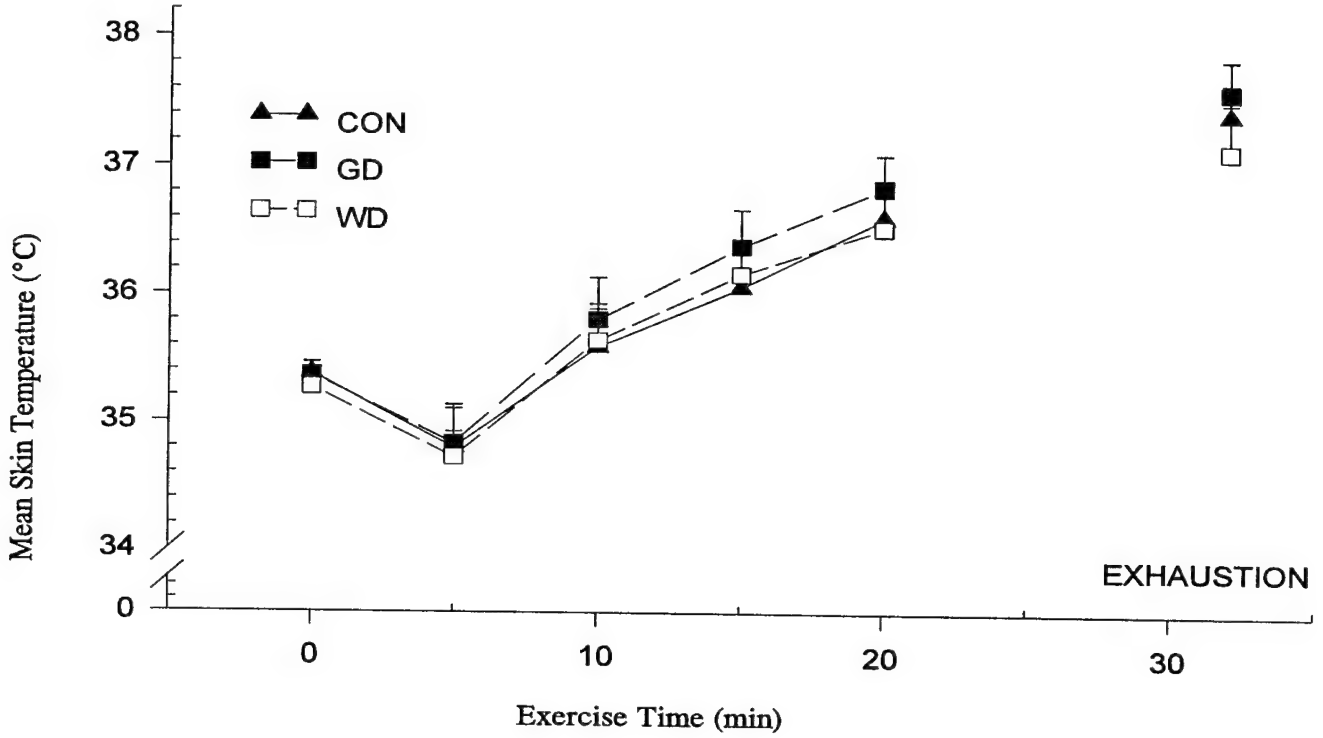

FIGURE 26: Mean skin temperature responses during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration.

FIGURE 27

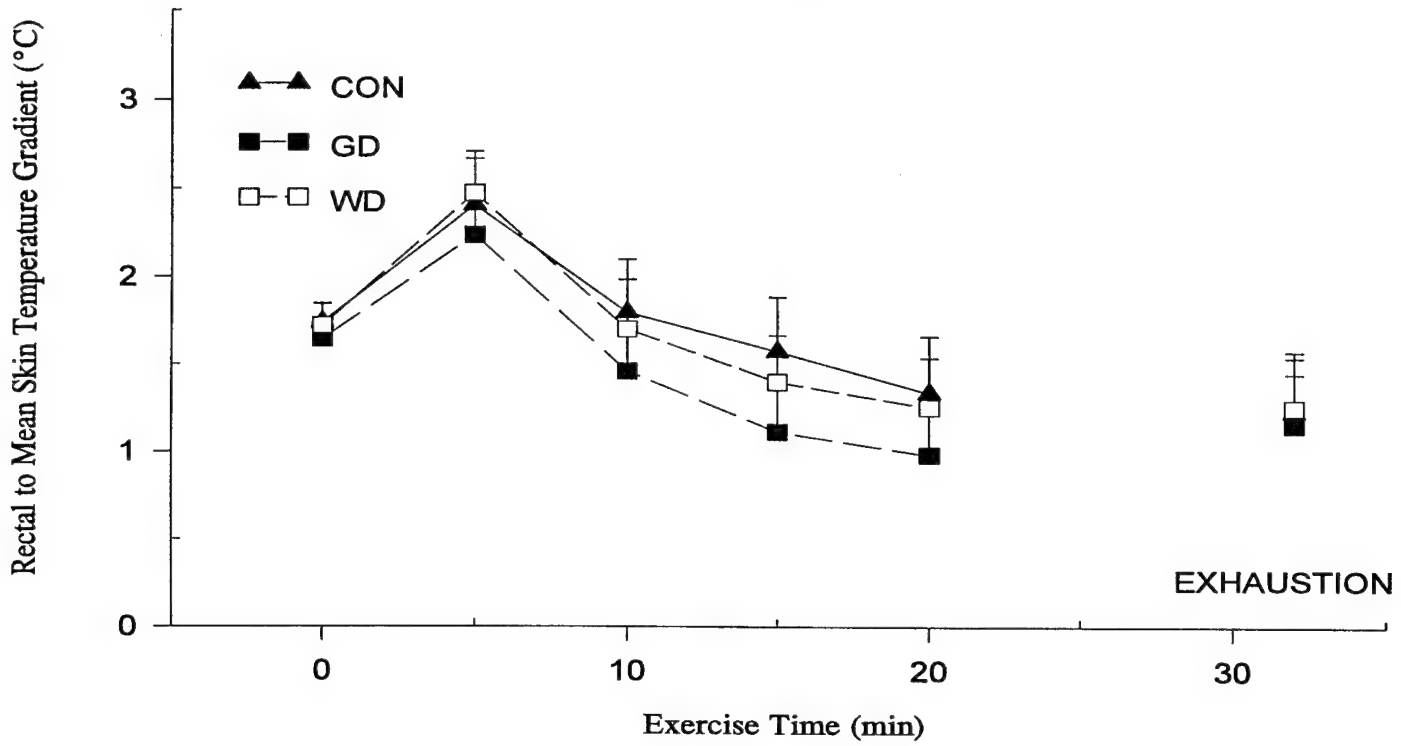

FIGURE 27: Rectal to mean skin temperature gradients during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration.

Figure 28 presents the mean body temperature ($\bar{T}_b$) responses during exercise for each trial. Mean body temperature values were not different ($P>0.05$) between trials and increased ($P<0.05$) over time during exercise. Final exercise $\bar{T}_b$ values were 38.5±0.4, 38.6±0.5 and 38.4±0.5 °C, for CON, GD and WD trials, respectively.

Figure 29 presents whole body sweating rate responses during exercise for each trial. Whole body sweating rates were not different ($P>0.05$) between trials. The whole body sweating rates were 608±119, 698±159 and 683±91 $g \cdot m^{2} \cdot h^{-1}$, for CON, GD and WD trials, respectively.

Physiologic Tolerance to Heat Strain

Table 9 presents the physiologic responses: rectal temperature, change in rectal temperature, mean skin temperature and heart rate measured at exhaustion from heat strain during CON, GD, and WD trials. At exhaustion, final T_{re}, change in T_{re}, final $\bar{T}_{sk}$, and final heart rate were similar $P>0.05$ in all trials. Final rectal temperature ranged from 37.9 to 39.7°C at exhaustion. Final change in T_{re} ranged from 0.9 to 2.4°C. Final $\bar{T}_{sk}$, ranged from 35.8 to 38.7°C. Final heart rate ranged from 162 to 204 $beats \cdot min^{-1}$ and heart rate values at exhaustion represent 95%, 95% and 94% of maximal heart rate for CON, GD and WD trials, respectively. **Figure 30** presents the individual's variability for core temperature tolerance during uncompensable exercise-heat stress. Four out of eight subjects had greater core temperature tolerance in the GD trials than the CON trials, and three out of eight subjects had greater core temperature tolerance in WD trials than CON trials.

Table 10 presents the frequency of symptoms reported at exhaustion relative to final temperature. At exhaustion, the reasons for discontinuing exercise included syncope / ataxia (n= 11), fatigue (n=10), apnea (n=2) and muscle cramps (n= 1). No apparent pattern was observed for frequency of symptoms relative to final rectal temperature.

FIGURE 28

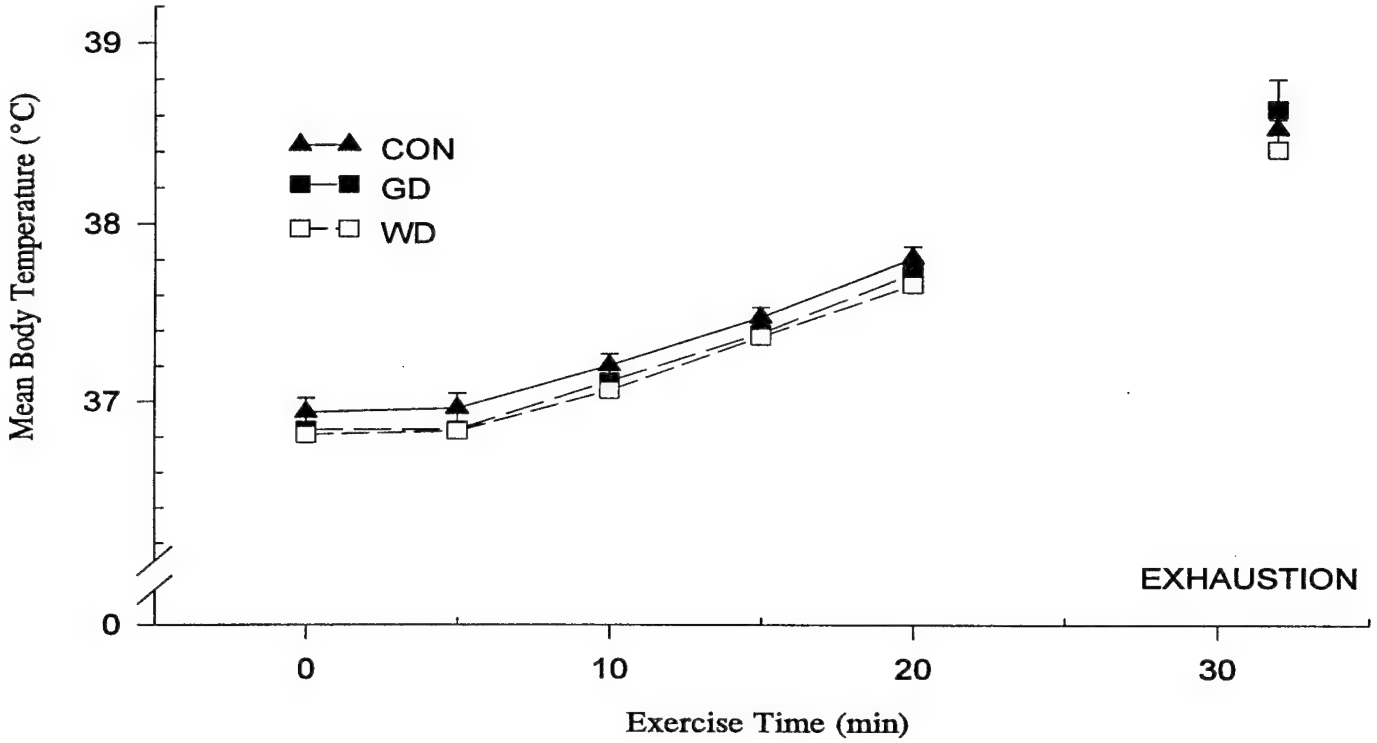

FIGURE 28: Mean body temperatures during uncompensable exercise-heat stress trials. Values are means ± SE; CON (filled triangles) = euhydration with no rehydration, GD (filled square) = glycerol hyperhydration with no rehydration, WD (open square) = water hyperhydration with no rehydration.

FIGURE 29

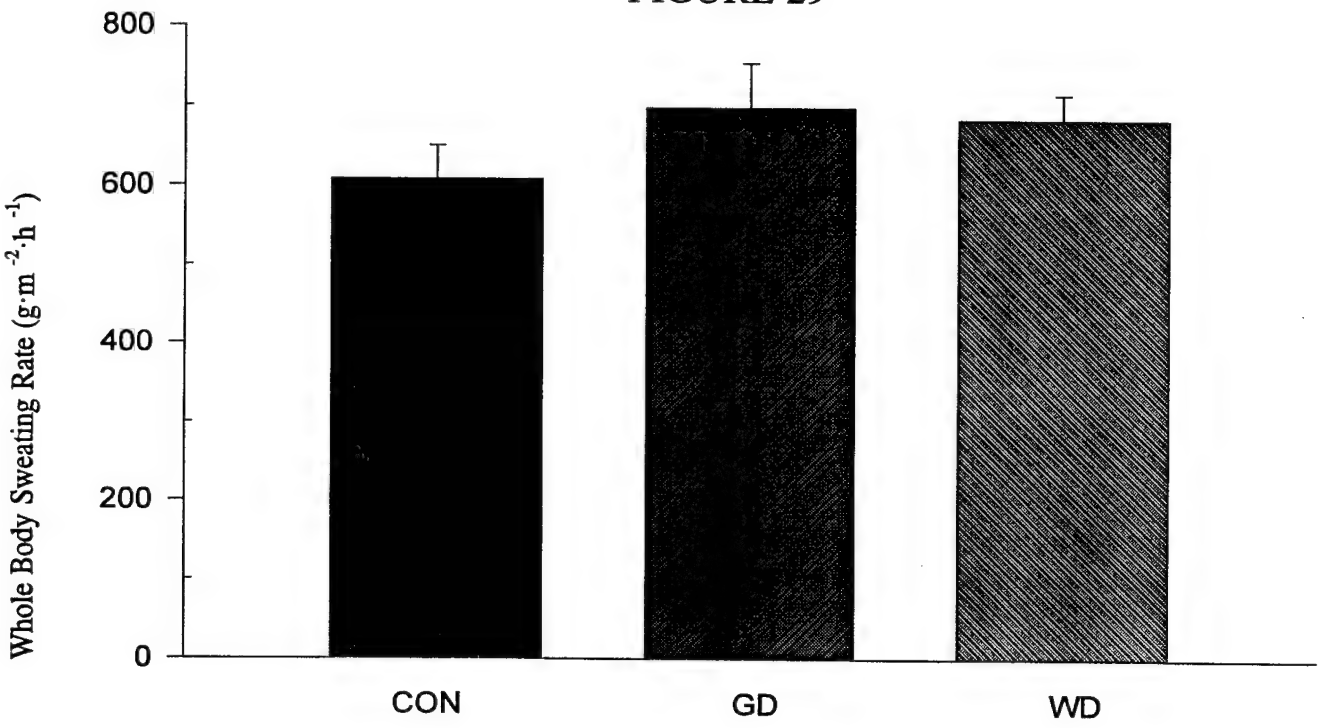

FIGURE 29: Whole body sweating rates during uncompensable exercise-heat stress trials. Values are means ± SE; CON = euhydration with no rehydration, GD = glycerol hyperhydration with no rehydration, WD = water hyperhydration with no rehydration.

FIGURE 30

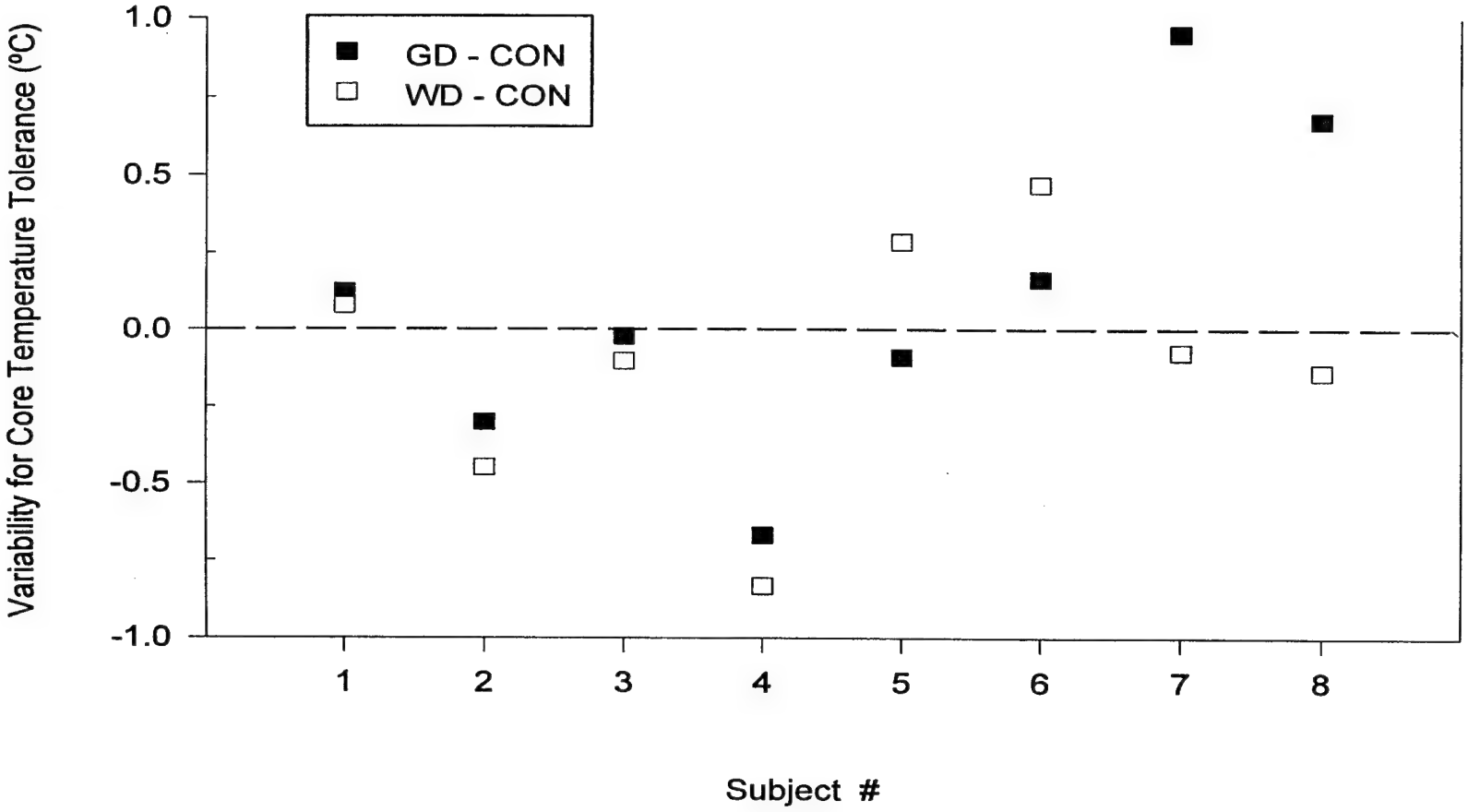

FIGURE 30: Individual's variability for core temperature tolerance during uncompensable exercise-heat stress trials. The values are GD and WD trials relative to CON trial, GD = glycerol hyperhydration with no rehydration, WD = water hyperhydration with no rehydration.

Table 9. Physiologic Responses at Exhaustion from Heat Strain for Control and Hyperhydration Trials During Uncompensable Exercise-heat Stress

Variable	Control	Glycerol Hyperhydration	Water Hyperhydration
Rectal Temperature (°C)	38.7±0.4	38.8±0.5	38.6±0.5
Rectal Temperature change, (°C)	1.5 ±0.4	1.7 ±0.4	1.6 ±0.5
Mean Skin Temperature (°C)	37.4±0.7	37.6±0.7	37.1±1.1
Heart Rate (beats·min^{-1})	187±12	187±8	185±13

Table 10. Frequency of Symptoms Reported at Exhaustion Relative to Final Rectal Temperature During Uncompensable Exercise - Heat Stress

Rectal Temperature (°C)	Syncope/ ataxia	Fatigue	Apnea	Muscle cramp
38.0		2		
38.2		2		1
38.4	2			
38.6	4	3	2	
38.8				
39.0	3	1		
39.2	1			
39.4		1		
39.6	1			
39.8		1		
Total	11	10	2	1

DISCUSSION

This study examined the efficacy of two hyperhydration approaches during compensable and uncompensable exercise-heat stress. A time schedule was used which initiated exercise-heat stress when TBW increases were expected to be greatest for both glycerol and water hyperhydration. In addition, the exercise-heat stress continued through the period (~90 min) when fluid retention differences between hyperhydration approaches were expected to be maximal (30). Therefore, the design should have been able to discriminate any initial and prolonged hydrational advantages between glycerol and water hyperhydration during exercise-heat stress.

A design emphasis in this study was to ensure that valid "baseline" conditions were maintained. Previous studies (35,42,69,75) reporting thermoregulatory advantages from hyperhydration have suffered from confounded baseline conditions where subjects were dehydrated. Therefore, those studies (35,42,69,75) most likely demonstrated the well-known adverse effects of hypohydration (65,66,97) rather than the possible advantages of hyperhydration. Different baseline conditions were selected for the present study's compensable and uncompensable heat stress trials. For the compensable heat stress trials, the baseline condition was maintained euhydration during exercise. This baseline condition was selected because during compensable heat stress, persons will maintain heat balance, thus exposure times can be prolonged with opportunities to rehydrate. For the uncompensable heat stress trials, the baseline condition was initial euhydration with subsequent progressive dehydration during exercise. This baseline condition was selected because during uncompensable heat stress persons will rapidly store heat (54,67,106), thus exposure times are short with few opportunities to rehydrate.

HYPERHYDRATION EFFECTS ON BODY FLUIDS

Both methods of hyperhydration were equally effective in increasing total body water. Total body water increased an average of 1.4 L and 1.5 L at 30 min post-drink during the compensable and uncompensable heat stress trials, respectively. Surprisingly, glycerol hyperhydration did not enable greater TBW than water

hyperhydration as reported by others (30,61,81). Studies (30,61,68,81) reporting greater TBW increases with glycerol than water hyperhydration were all on resting subjects in temperate climatic conditions; however, not all studies comparing glycerol and water hyperhydration have reported greater TBW increases with glycerol (6,68). Montner et al.(68) used identical hyperhydration procedures in two studies, and reported that at 1-hr post-drink (resting in temperate climate) glycerol induced greater TBW increases in one study, but not in the other study.

Freund and colleagues (30) recently published a comprehensive paper regarding glycerol hyperhydration and the mechanisms of action. That study employed hyperhydration methodologies identical to those in this study. In agreement with Freund et al. (30), the TBW increase was similar for both hyperhydration approaches at 30 min post-drink, even though our subjects were resting in a warm (35°C) climate. This study was unable to confirm Freund et al.'s (30) observation of a greater TBW increase with glycerol than water hyperhydration after 90 min post-drink; however, from 30 min to 180 min post-drink our subjects were performing exercise in the heat, while Freund's subjects were resting in a temperate climate (30). Previous studies (61,68) that compared glycerol to water hyperhydration during exercise have not shown any TBW advantages with glycerol. Lyons et al. (61) reported a greater TBW increase ~0.5 L at 120 min post-drink with glycerol hyperhydration while resting in a temperate climate, but this advantage was not observed after exercise in heat. Likewise, Montner et al. (68) reported no advantage in TBW increase with glycerol hyperhydration (compared to water) during exercise in a temperate climate.

Freund and colleagues (30) found that the greater TBW increase with glycerol hyperhydration was associated with lower free water clearance (compared to water hyperhydration). They stated that slightly higher ADH levels may have contributed to the lower free water clearance. Freund and colleagues (30) proposed that the primary mechanism mediating a greater TBW increase with glycerol hyperhydration (compared to water) was its direct effect on the kidney, independent of any hormonal responses. They (30) proposed that glycerol loading might cause an increased renal medullary osmotic concentration gradient and thereby decrease free water clearance. Glycerol could increase renal medullary osmotic concentration gradient by either direct reabsorption of glycerol in the distal tubule (115), or by indirectly increasing Na^+ reabsorption in the distal tubules (30). An increased medullary osmotic concentration

gradient should increase free water reabsorption and, therefore, decrease free water clearance and reduce diuresis (30).

An important question is why did glycerol hyperhydration provide no additional TBW increase during exercise-heat stress? Exercise-heat stress should decrease renal blood flow (18,49,123), decrease glomerular filtration (18), decrease free water clearance (18), as well as reduce diuresis (18,123). This was apparent as urine flow decreased by ~70% (5.4±3.6 to 1.7±1.2 $ml \cdot min^{-1}$) from rest to exercise and was not different between hyperhydration trials. Therefore, the effectiveness of exercise and heat on reducing diuresis probably outweighed the effects of glycerol on decreasing free water clearance.

We found that hyperhydration did not elicit a plasma volume expansion during either rest or exercise in the heat. Previous investigators report disparate results regarding plasma volume expansion with glycerol hyperhydration during rest and exercise. Glycerol hyperhydration has been reported to expand plasma volume during rest (37) and exercise (70) in temperate climates, while other investigators reported no differences during rest in temperate climates (81) or during exercise in heat (61). Freund and colleagues (30) reported that glycerol and water hyperhydration both expanded plasma volume (by ~150-200 ml) during rest in a temperate climate. Plasma volume represents only ~8% of TBW (96), so if a 1.5 L TBW increase is equally distributed throughout body water compartments, then plasma volume should only expand by ~120 ml. This small plasma volume expansion is likely at the threshold of measurement resolution and any other perturbation could mask it. In this study, subjects were exposed to exercise and additional heat stress between blood draws. Exercise-heat stress will induce large changes in plasma volume (96) that can easily mask any subtle effects expected from hyperhydration.

COMPENSABLE EXERCISE-HEAT STRESS

The compensable heat stress trials were designed so that heat loss would primarily occur by evaporation (33,105). This should maximize core temperature differences from any sweating rate advantage incurred by hyperhydration, and previous studies have suggested that hyperhydration might increase sweating rates (see

Background section on Hyperhydration). The exercise intensity and climatic conditions were selected so that heat balance could be maintained and steady-state core temperature responses could be achieved. The E_{req} was 293 $W \cdot m^{-2}$ and the E_{max} was 462 $W \cdot m^{-2}$, thus the E_{req}/E_{max} was 63%.

This study's data clearly demonstrate that hyperhydration provides no physiologic advantage compared to euhydration during compensable exercise-heat stress. Compared to euhydration, hyperhydration did not modify rectal temperature, esophageal temperature, skin temperature, local sweating, whole body sweating or heart rate responses. In addition, glycerol hyperhydration provides no physiologic advantage compared to water hyperhydration, as responses were essentially identical for both sets of trials. These findings support the notion that previous studies demonstrating thermoregulatory advantages with hyperhydration may have simply demonstrated the adverse effects of hypohydration, or had results systematically confounded from inadequate experimental designs (e.g., treatment order effect causing heat acclimation).

Recent interest in glycerol hyperhydration to improve exercise-heat performance originated from the study of Lyons and colleagues (61). They reported that glycerol hyperhydration induced remarkable core temperature (~0.7°C) reductions and sweating rate (~0.3 $L \cdot h^{-1}$) increases, with no effects on heart rate during compensable heat stress; that study also reported that water hyperhydration provided no physiologic advantage compared to control. The primary difference between studies was that Lyons et al. (61) used subjects who were unfit (42 $ml \cdot kg^{-1} \cdot min^{-1}$, $\dot{V}O_{2\ max}$), unacclimated to heat, and who performed higher intensity exercise (60% $\dot{V}O_{2\ max}$) at a higher E_{req} / E_{max} (~77%). It might be argued that glycerol hyperhydration provided a greater advantage in their unacclimated subjects and, therefore, these effects would not be present in heat acclimated subjects, as used in this study. If that were true, then from a practical point of view, it would be more effective to heat acclimate persons than to glycerol hyperhydrate them. Most adaptations to heat acclimation can be accomplished within four days of heat exposure (23,105), and acclimation provides a physiologic advantage for extended durations, without the side effects of hyperhydration (urine output, gastric distress, headaches, etc.).

Time-line differences may have contributed to disparate results between studies. Lyons and colleagues (61) initiated exercise at 150 min (vs. 60 min in this study) post-glycerol ingestion when peak TBW differences (~0.5 L) were observed between hyperhydration approaches. However, in the present study, there was a ~1.5 L TBW increase with hyperhydration and no observed physiologic advantages. Montner et al. (68) used a similar time-line as Lyons et al. (61) and reported no differences in core temperatures and sweating rates between glycerol hyperhydration and water hyperhydration trials during exercise (~75% $\dot{V}O_{2\ max}$) in a temperate climate. Another argument might be that glycerol provides a thermoregulatory effect (irrespective of TBW changes), because Lyons et al. (61) showed physiologic advantages with glycerol hyperhydration but not with water hyperhydration. The present study used essentially the same glycerol dosage as those investigators and found no evidence for any physiologic advantage.

The sweating threshold and sensitivity values reported here are similar to those reported for euhydrated, heat-acclimated subjects using identical methodology (66,99). Research has demonstrated that changes in blood volume and tonicity can alter the control of sweating (for review, see reference (97)). In this study, hyperhydration did not alter blood (plasma) volume; however, large increases in serum osmolality with no changes in serum sodium were observed. In the glycerol hyperhydration trials, serum osmolality increased because of the glycerol load and not because of increased sodium as in other thermoregulatory studies. The observed increase in serum glycerol concentration (by ~120 $mg \cdot dl^{-1}$) can fully account for the 13 $mOsmol \cdot kg^{-1}$ serum osmolality increase. Glycerol should penetrate osmosensitive cells within the thermoregulatory centers, so any osmotic increase from this might not be expected to alter control of thermoregulation (110).

Hyperhydration delayed the achievement of a body water deficit during trials when fluid was not replaced. Figure 1 illustrates that TBW was below euhydration levels after ~60 min of exercise when fluid was not replaced during hyperhydration trials. As expected, when hypohydration was present, physiologic strain (esophageal temperature, heart rate) was increased. However, we did not observe additional rectal temperature increase when fluids were not replaced. The difference between esophageal and rectal temperature responses might be explained by the response time of these measurements. The response time for esophageal temperature to reflect

changes in blood temperature is about 20 min, whereas the response time for rectal temperature to reflect changes in blood temperature may be up to 40 min (104).

UNCOMPENSABLE EXERCISE-HEAT STRESS

The uncompensable heat stress trials were designed to focus on potential cardiovascular and tolerance advantages from hyperhydration, as the exercise-climatic-clothing conditions would not allow heat balance. The E_{req} was 366 $W \cdot m^{-2}$ and E_{max} was 88 $W \cdot m^{-2}$, so the E_{req} / E_{max} was 416%. During all trials, subjects continually stored body heat until exhaustion occurred. The subjects were highly motivated and no doubt existed regarding the presence of exhaustion in all cases. The reasons for discontinuing exercise included syncope/ataxia (n= 11), fatigue (n=10), apnea (n=2) and muscle cramps (n= 1). Endurance time averaged 32 min for all trials and ranged from 24 to 38 min.

This study was the first to examine the efficacy of hyperhydration for uncompensable heat stress. The data clearly demonstrate that glycerol (compared to water) hyperhydration provides no physiological or tolerance advantage during uncompensable exercise-heat stress. Compared to water hyperhydration, glycerol hyperhydration did not modify rectal temperature, skin temperature, whole body sweating, mean arterial pressure, total peripheral resistance, cardiac output, heart rate responses, nor endurance time or physiologic strain at exhaustion during uncompensable exercise-heat stress.

Compared to control conditions (euhydration with no fluid replacement), hyperhydration demonstrated several minor advantages. Glycerol hyperhydration increased endurance time (~4 min), lowered heart rate (~10 beats·min^{-1}), while water hyperhydration lowered heart rate (~10 beats·min^{-1}) relative to control. These advantages probably reflect the adverse effects of hypohydration during the control trial. By the end of the control trial, subjects were hypohydrated by ~1.5 L (<2% of body weight). Hypohydration of this magnitude will increase physiologic strain during compensable exercise-heat stress (65,78,109,114), however, its effects on short-term uncompensable heat stress are not well studied (106). Sawka and colleagues (106) studied hypohydration (~5% of body weight) during uncompensable heat stress, but

they only reported physiologic responses at exhaustion. In this study, the combination of short exposure time, delay until significant hypohydration developed, and delays between hypohydration occurring and mediating increased physiologic strain probably all contributed to the adverse effects of hypohydration not being more evident.

Hyperhydration did not alter the physiologic strain tolerated at exhaustion during uncompensable exercise-heat stress. Exhaustion from heat strain occurred at the same core temperature, core temperature increase, heart rate and skin temperature during each trial (see **Table 9**). Previous research has demonstrated that core temperature provides the "best" physiologic index of thermal tolerance (67,74,106). In the present study exhaustion from heat strain occurred at a core temperature of 38.7±0.5°C, this compares favorably to values reported by other studies examining uncompensable heat stress (67,74,106). Montain et al. (67) reported that exhaustion from heat strain occurred at a core (rectal) temperature of 38.6±0.5°C for subjects wearing protective clothing in variety of different exercise intensities and climatic conditions (all uncompensable heat stress). Sawka et al. (106) reported that exhaustion from heat strain occurred at a core (rectal) temperature of 39.1±0.3°C for euhydrated, and 38.7±0.7°C for hypohydrated subjects who only wore shorts. Nielson et al. (74) reported that exhaustion from heat strain occurred at a core (esophageal) temperature of 39.7±0.2°C for subjects who only wore shorts.

The research approach used in this study should have had sufficient resolution to observe any meaningful hyperhydration effects on physiologic tolerance to uncompensable heat stress. Using the same research approach, physiologic tolerance to uncompensable heat stress has been reduced by both hypohydration (106) and by wearing protective clothing (67). Reductions of core temperature tolerance by ~0.4°C and ~0.3°C were induced with hypohydration and wearing protective clothing, respectively. Hypohydration was thought to reduce physiologic tolerance to heat strain because of the decreased blood volume resulting in less cardiac filling (105,106). Wearing of protective clothing was thought to reduce physiologic tolerance to heat strain, because of the higher skin temperatures causing greater blood displacement from the central circulation to the skin and resulting in less cardiac filling (12,67). Clearly, reductions in cardiac filling are believed to be responsible for differences in physiologic tolerance to uncompensable heat stress.

During exercise-heat stress, the simultaneous dilation of cutaneous and skeletal muscle vascular beds is associated with reduced cardiac filling and stroke volume (71,90,105). During severe exercise-heat stress, cardiac filling can decline to a level where cardiac output cannot be maintained (87,90) by increasing heart rate. Rowell et al. (92) reported that during uncompensable exercise-heat stress, cardiac output can be reduced by ~1 L/min compared to control values measured in a temperate climate. In this study, cardiac output decreased (1 L/min), stroke volume decreased (14 ml) and heart rate increased (14 beats·min^{-1}), which all indicate that cardiac filling was declining. It was anticipated that hyperhydration would increase blood volume (30,68,97), thereby allowing improved cardiac filling and cardiac output to be better maintained (compared to control). Based upon the compensable heat stress data, it is unlikely that hyperhydration increased blood volume during the uncompensable heat stress trials. It is unclear, however, why hyperhydration mediated slightly lower heart rates with no other cardiovascular or heat tolerance advantages.

Physiologic mechanism(s) suggested being responsible for exhaustion from heat strain (13) include the following: high circulatory strain (88), reduced motivation from high body temperatures (13), increased discomfort from hot/wet skin (39,67), reduced blood flow to skeletal muscle causing hypoxia (24,43), increased muscle anaerobic metabolism and glycogen utilization (25,122), and reduced blood flow to brain causing syncope (49). It is possible that any combinations of these factors were responsible for exhaustion from heat strain in this study. As discussed above, there was some evidence that hyperhydration reduced circulatory strain with no heat tolerance advantage. Therefore, cardiovascular strain was not the sole reason for exhaustion. Numerous subjects discontinued exercise at relatively low body temperatures, as also reported by others (54,67); therefore, the effects of high body temperature on motivation were not the sole reason. Exhaustion occurred after a relatively short period for this exercise intensity, so it is doubtful that differences in glycogen depletion were an important factor for exhaustion in this study. The high incidence of reported syncope/ataxia (11 of 24 cases) at exhaustion suggests that cardiovascular system may have contributed to exhaustion in many instances. In summary, these physiologic mechanisms responsible for heat exhaustion may be integrated so that their combined actions could account for a reduction in motivation to continue exercise, (13) or if

motivation is maintained, exercise could continue until physiological limitations are incurred (e.g., ataxia/syncope).

PRACTICAL CONSIDERATIONS FOR HYPERHYDRATION

Hyperhydration has adverse effects (increase urine flow, nausea, headaches, etc.) that could limit its application for persons working in hot climates. The nausea and headache from glycerol consumption are documented (70,118), but the incidences of these adverse reactions have not been documented by previous glycerol hyperhydration studies (68,81). Murray et al. (70) and Tourtellote (118) have reported adverse reactions from glycerol solution ingestion which included nausea and headaches. Those investigators did not report the incidence or severity of these adverse reactions. Other studies (30,61,68,81) have not documented any adverse reactions from glycerol solutions ingestion.

In this study, three of nine subjects (total of five trials) became nauseous from ingesting the glycerol solutions, so these trials were terminated and repeated another day. One subject, however, repeatedly became nauseous from drinking glycerol solution and was removed from the study. The adverse reactions may have been in-part aggravated by the large volume of warm water consumed after the glycerol bolus. In this study, no subjects reported headaches during exercise, but two of eight subjects (total of four trials) reported headaches after the exercise in glycerol trials. Glycerol induced headaches could easily have been overlooked in double-blind studies in which the symptoms occurred after the trial and subjects had left the laboratory. In summary, individual tolerance to glycerol ingestion varies daily, and it appears certain subjects are more prone than others.

Glycerol has recently been commercially marketed as a nutritional aid to help athletes prevent hypohydration, and has recently been made available in stores (3). The commercial products use essentially the same dose as employed in this study. The commercial marketing of glycerol as a nutritional aid is not scientifically accurate. In a brief article on glycerol supplement in the magazine Outside, Dr. Montner was quoted "Glycerol helps the body retain water and slows dehydration...glycerol acts like a sponge, storing the water you need for healthy perspiration and circulation (3)." In the

present study, glycerol hyperhydration provided no hydrational or physiologic advantage compared to water hyperhydration; in addition, hyperhydration provided no physiologic advantage over maintaining euhydration.

CONCLUSIONS

This study examined the efficacy of hyperhydration approaches during compensable and uncompensable exercise-heat stress and the impact of hyperhydration on physiological tolerance to heat strain. It is concluded that hyperhydration provides no meaningful advantages over the maintenance of euhydration during exercise-heat stress. This conclusion is based upon the following observations:

1) Glycerol solution ingestion can result in nausea and headaches, and the response varies between persons.

2) Glycerol hyperhydration provided no hydrational advantage over water hyperhydration during exercise-heat stress. Both hyperhydration approaches increased TBW by similar amounts.

3) During compensable exercise-heat stress, thermoregulatory responses were nearly identical regardless of whether subjects were euhydrated, water hyperhydrated or glycerol hyperhydrated.

4) During uncompensable exercise-heat stress, physiologic strain and heat tolerance were nearly identical regardless of whether subjects were water hyperhydrated or glycerol hyperhydrated.

5) Hyperhydration delayed the development of body water deficits if fluids were not replaced during exercise-heat stress.

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